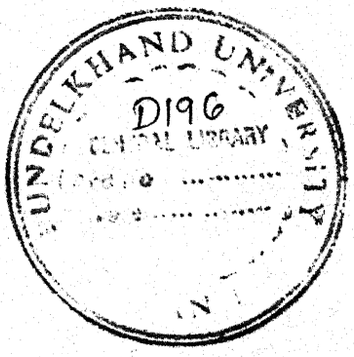
CLINICAL PROFILE OF LOW BODY WEIGHT TYPE-2 DIABETES MELLITUS IN BUNDELKHAND REGION

THESIS FOR

DOCTOR OF MEDICINE

(INTERNAL MEDICINE)



BUNDELKHAND UNIVERSITY
JHANSI (U.P.)

2003 SUSHIL KUMAR MISHRA

Dedicated

To

Respected

Teachers, Parents,

Friends & my Supportive

Family members

Acknowledgement

Though we thank the Supreme Power daily but I feel deeply blessed when I see my hard work and the blessings of Supreme Almighty and my elders materializing.

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About my Co-Guide ***Prof. (Dr.) R.C.Arora*** *M.D, D.Sc. Principal,Professor and Dean Department of Medicine, M.L.B. Medical College, Jhansi, the more said is less. His affectionate guidance, invaluable support and keen interest have helped me immensely. The constant encouragement and subtle criticism offered by him were the source of strength that enabled me to trod forward over the obstacles.*

I am extremely thankful to Co-Guide ***Dr. Mrs. Sunita Arora*** *M.S., Professor, Department of Obstetrics and Gynaecology, M.L.B. Medical College, Jhansi, whose continuous supervision and assistance helped me a lot*

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Lastly but not the least I am thankful to all the patients who were the very basis of this study.

Dated: 7/9/03

Sushil Kumar Mishra.

CERTIFICATE

This is to certify that the work entitled ***"Clinical profile of low body weight Type-2 Diabetes Mellitus in Bundelkhand region"*** which is being submitted as a thesis for M.D. (Medicine) Examination 2003 of Bundelkhand University, Jhansi, has been carried out by ***Dr.Sushil Kumar Mishra*** in the Department of Medicine, M.L.B. Medical College, Jhansi.

This method described was undertaken by the candidate himself and the observations recorded have been periodically checked up. He has put in the necessary stay in the Department as per University regulations, and has fulfilled the conditions required for the submission of thesis according to University regulations.

Dated: 7 / 7 /03

Dr. R.C Arora
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CERTIFICATE

This is to certify that the work entitled ***"Clinical profile of low body weight Type-2 Diabetes Mellitus in Bundelkhand region"*** which is being submitted as a thesis for M.D. (Medicine) Examination 2003 of Bundelkhand University, Jhansi, has been carried out by ***Dr.Sushil Kumar Mishra*** under my direct supervision and guidance, the techniques embodied in the thesis were undertaken by the candidate himself and the observations recorded have been checked and verified by me from time to time.

Dated: / /

Dr. Navnit Agarwal
M.D.,
Professor Department of Medicine,
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Dated: 7/07/03

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Introduction

Introduction

Diabetes was described more than 2000 years ago. For the past 200 years it has featured in history of modern medicine[1]. As we enter the new millenium, diabetes has become a problem of epidemic proportion. It touches us all in every walk of life – Physicians and Scientists, Family and Friends, even Government and Communities- and it imparts a constantly toll. Over Society as a whole more and more people have problems requiring answers; and in these days of rapidly expanding technology they find it difficult to shift through all the information[2]. Today, more than ever, "to know diabetes is to know medicine".

Diabetes mellitus is the most prevalent non-communicable group of common metabolic disorders that share the phenotype of hyperglycemia in the world. Several distinct types of DM exist and are caused by a complex interaction of genetics, environmental factors, and life-style choices. Depending on the etiology of the DM, factors contributing to the hyperglycemia may include reduced insulin secretion, decrease glucose usage, and increased glucose production. The metabolic dysregulation associated with DM cause secondary pathophysiologic changes in multiple organ systems, that impose a tremendous burden on the individual with diabetes and the health care system. With increasing incidence worldwide, DM will likely continue to be a leading cause of morbidity and mortality in the forthcoming future.

Recent changes in classification[3,4,5,6,7] reflect an effort to classify DM on the basis of pathogenic process that lead to hyperglycemia as opposed to criteria such as age of onset or type of therapy.

Etiological classification of diabetes Mellitus

1. Type 1 diabetes (β cell destruction, usually leading to absolute Insulin deficiency)

I_A Immune mediated

I_B Idiopathic

2. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance).

3. Other specific type of diabetes

3_A Genetic defects of β cell function characterized by mutation in :-

1. Hepatocyte nuclear transcription factor (HNF).
   4α -(MODY-1)
2. Glucokinase (MODY-2)
3. HNF 1α (MODY-3)
4. Insulin Promoter Factor (IPF)-1 (MODY-4)
5. HNF1β (MODY-5)
6. Mitochondrial DNA
7. Proinsulin or insulin conversion

3_B Genetic defect in insulin action

1. Type A insulin resistance
2. Leprechaunism
3. Rabson Mendenhall syndrome
4. Lipoatropic diabetes

3_C *Disease of the exocrine pancrease*

(Pancreatitis, Pancreatectomy, neoplasia, Cystic fibrosis, hemo chromatosis)

3_D *Endocrinopathies*

(Acromegaly, Cushing syndrome, hyperthyroidism, pheochromo cytoma, aldosteronoma)

3_E *Drug or chemical induced*

(Glucocorticoid, pentamidine, nicotinic acid, thyroid hormone, β adrenergic agonists, thiazides, phenytoin, α Interferon, β Blockers).

3_F *Infection- Congenital rubella, CMV, Coxsackie*

3_G *Uncommon form of immune mediated diabetes*

(Stiffman syndrome, Anti insulin receptor antibodies)

3_H *Other genetic syndromes*

(Down's, Klinefelter, Turner's, Friedrich's ataxia, Huntington's Chorea, myotonic dystrophy, porphyria, Prader Willi syndrome)

4. Gestational diabetes mellitus (GDM)

By such nosology a huge group approximately 150 million people (nearly 90% of diabetic population) emerged a Type 2 Diabetes Mellitus which is the commonest form of Diabetes Mellitus seen all over the world; and by itself is highly heterogeneous in patients profile manifestation, complication and management. The prevalence of Type 2 diabetes varies in different geographic regions and different ethnic group. Prevalence of Type 2 diabetes is increasing in most of the countries, especially in developing countries such as India.

According to the recent WHO reports, the prevalence of diabetes mellitus in adults worldwide will rise from 4% in 1995 to 5.4% in the year 2025 and more than 75% of them will be residing in developing countries[8,9].

Diabetes in India

Diabetes mellitus has emerged as a major public health problem in our country and has assumed epidemic proportion. According to Indian Council of Medical Research – Prevalence of diabetes mellitus has increased from 2.1% in 1972 to almost 12% in 1995. The vast majority of diabetes in India are Type 2. Prevalence of Type 2 DM has changed from 1972 2.3% in urban and 1.5% in the rural areas to 11.5% in urban and 2.4% in rural areas. WHO recognized two sub clinical type of Type 2 as obese and non-obese[10,11,12,13]. In contrast to WHO prediction of 60-80% to be obese only, about 30% of Indian diabetics are obese and vast majority of them were found to be non-obese. The age of onset is a decade earlier from their counterparts in the developed countries[17,19,48,49].

There is a sub type of Type 2 diabetics, who are lean and underweight (below 20% of the expected ideal body weight for height) with a body mass index (B.M.I) < 18.5 Kg/m^2 . They are different from patients who loose weight at the onset of diabetes. Attention to this specific type of underweight Type 2 diabetes was first drawn by Tripathi and Kar[14] in 1965, who reported that 28% of their adult onset diabetics were underweight and they did not develop Ketoacidosis. However, they thought that this was because of under nutrition.

Das in 1991 highlighted these clinical features, biochemical, hormonal profiles which confirmed that there are variants of Type 2 diabetes.

Therefore, in the past these adult onset diabetics were variously nomenclatured as underweight, undernourished, lean and even non-obese by different authors. However, the International workshop on "Types of diabetes peculiar to tropics", held in Cuttack[15,16] gave these diabetics defined nomenclature of low body weight as they were neither malnourished nor the loss of weight could be attributed to the prevalent metabolic state.

However, the various studies done at various centers in India (such as Calcutta, Cuttack, Chennai, Hyderabad, Madurai & Jaipur) have showed that these low body weight type-2 diabetics revealed significant difference in their clinical, biochemical, hormonal and mortality profile.

Since most of these studies on low body weight type-2 diabetes mellitus done in our country are from other states. No study has been done in our state U.P. and Bundelkhand region as well as in Department of Medicine Jhansi.

This very thought enriched us with new enthusiasm and we planned to study the clinical profile of low body weight type-2 diabetes mellitus in Bundelkhand Region in the Department of Medicine, Maharani Laxmi Bai Medical College, Jhansi, U.P.,India.

Essentials of diagnosis of diabetes[7] :

1. *Symptom of diabetes plus random blood glucose concentration ≥200 mg/dl (11.1mmol/L)*

OR

2. *Fasting (no caloric intake for atleast 8 hr) plasma glucose ≥126mg/dl (7 mmol/L)*

OR

3. *Two hour plasma glucose ≥200mg/dl (11.1 mmol/L) after 75 gm oral glucose load.*

Etiological determinants and risk factors for type-2 diabetes :-

1. *Genetic factor*

(a) Genetic marker (b) Family History (c) Thrifty gene(s) etc.

2. *Demographic characteristics*

(a) Sex (b) Age > 45 yrs (c) Pace/Ethnicity

3. *Modifiable risk factor (Including behavioural and lifestyle related)*

- Obesity (including distribution & duration)
- Physical inability
- Diet
- Westernisation, Urbanisation, and Modernisation
- Intra Uterine environment

4. *Other*

- Hypertension ≥ 140/9 mm Hg
- HDL ≤ 35 mg/dl Triglyceride > 250 mg
- History of GDM

Review Of Literature

Review of Literature

Low body weight Type-2 DM is clinical subtype of Type-2 DM and is characterized by low body weight i.e., BMI 18.5 Kg/m^2. They manifest with visible different presentation, morbidity & mortality pattern as well as biochemical profile when compared with classical patient of Type-2 DM.

The onset of diabetes is insidious with varieties of somatic complaints and loss of general well being. All are not poor and some belong to upper middle class society. Leanness is an inherent characteristics of these individuals. Good metabolic control had little influence on their constitution, natural history or morbidity. These diabetics in due course, more often become insulin require as compared to classical Type-2 DM patients[17,18,19,20,21,22,23]. These clinical characteristics biochemical profile, hormonal status and other features has been assessed by different worker as.

1. Tripathi BB and Kar B.C[14]

Tripathi and Kar in their premium publication on clinical type of diabetes in India way back in 1965, had reported that as high as 28 percent of adult onset diabetics were underweight. The said article had focussed on the relationship of malnutrition and presentation of diabetes, more so in the young with special emphasis on malnutrition related diabetes mellitus. Thus for past these adult onset diabetics were variously nomenclatured as under weight, under nourished, lean and even non-obese by different authors.

However, the International workshop[15,16] on type of diabetes peculiar to tropics gave these diabetics the defined nomenclature of low body weight as they were neither malnourished nor the loss of weight could be attributed to the prevalent metabolic state[20].

2. A.K. Baliarsinha and Siddarth Das[13,23,24]

On the basis of their study as 380 patient of Type-2 DM recruited consecutively, 91 (23%) had BMI $\leq$ 18.5 kg/m^2 and fall over a period of two consecutive years.

They observed that more than 80% of patient with LB Type-2 DM were from the middle socio-economic class. In India middle class constitutes about 50% of the population and these type of Type-2 DM was probably the real representation of DM prevalent in such society.

Family history of DM could be ascertained in 16.5% of such patients which is much higher than any form of malnutrition related diabetes mellitus of Type-1 DM.

LB Type-2 diabetics had much lower WHR and there was strong positive correlation between BMI and WHR suggesting that leanness as well as levels of central obesity were expression of a common underlying mechanism.

These LB Type-2 diabetic patients presented with long standing Hyperglycemia without ketosis suggesting endogenous insulin reserve that prevent ketonebody production but are unable to prevent hepatic glucose suppression.

In these series PN was the commonest presenting feature followed by Tuberculosis, Skin or Fungal infection. On the other hand hypertension, CAD weir less common in comparison to obese or standard weight Type-2 DM.

Glycemic status in majority of LB Type-2 DM patient (80.5%) were controlled with OHA and only few (16%) required insulin or insulin & OHA both (4.5%). Initial response to OHA in majority of cases, indirectly denote that the LB Type-2 DM patient had good β cell reserve and lack of insulin resistance. Both cholesterol & tri glycerides were low or normal with a higher HDL values which could contribute to lower incidence of macro vascular disease in LB Type-2 DM.

The basal IRI values in LB Type-2 DM, despite more severe hyper glycemia was significantly lower than obese Type-2 indicates that the LB patient are more insulinopenic or had decreased beta cell reserve.

The most significant finding of this study was that, although the basal, post glucose and post glucagon values of IRI were significantly lower in the LB Type-2 DM there was no statistical difference in the corresponding C-peptide levels. Thus the β cell reserves of insulin and its response to secretogogues, both glucose and non-glucose, were identical in their two group which testified that they are classical Type-2 diabetic patient. Yet gross disparity in the C-peptide versus IRI levels in the peripheral circulation requires explanation. The most plausible mechanism is inappropriate extraction of insulin by the liver in the LB compared to obese Type-2 DM[25].

3. Anant Nigam[26]

This study was carried out at a diabetes center in NorthWest India, the 1689 consecutively registered Type-2 DM patient 167 (9.8%) of LB Type-2. The majority of them had onset of disease at age of 30 yr as after had no ketone urea at diagnosis and did not

need insulin treatment at diagnosis. Of these 27% of LB Type-2 DM patient were having central obesity as shown by increased WHR, suggested that central obesity is also probably a pointer that these patients are group of Type-2 DM patient[27].

4. Mohan et al[10,12,28]

This study was conducted in a sub set of low body weight Type-2 DM for presence of GADA and ICA. GADA were present in 10% of low body weight patients as compared to a frequency of 5% in normal weight and 4% in obese Type-2 diabetics. The frequency of ICA was 0% in low body weight 13% in normal weight and 7% in obese diabetics respectively. This data suggest that islets antibody were not more frequent in low body weight Type-2 DM compared to diabetics with higher BMI.

5. Ram Chandran et al[29,30]

They had revealed that GADA have low positivity and lower sensitivity with regard to prediction for insulin requirement in Indian Type-2 diabetics. The prevalence of autoantibodies in Type-2 DM was low so also its predictability to determine insulin requiring state in such diabetics was statistically of no consequence.

6. K. Kanan and A.J Arirratham[11,31,32]

In this study peak C-peptide response measured in the small groups of Type-2 and Type-1 subjects revealed typical finding which are the basis of categorization of LB Type-2 subject as distinct entity.

LB Type-2 diabetics did not have much insulin resistance but C-peptide output was the least of the three categories but much higher than Type-1 diabetes.

They finally concluded that the absence of any strong family tendency and a strong male preponderance of cases make it possible that LB Type-2 DM is in same way different from the other subjects with Type-2 disease. The low incidence of hypertension and severe dyslipidemia can also be explained by low BMI, and absence of hyperinsulinemia in these diabetics, ECG and clinical symptoms of CHD is low at 12%.

The absence of evidence for any degree of insulin resistance in these diabetics conveys a message that biguanides and/or other insulin sensitizers are not going to have much therapeutic advantage in them. It is unlikely that chronic infective condition like tuberculosis might have contributed to the loss of weight as the incidence of tuberculosis is lower than what is seen in other Type-2 diabetics.

7. Alok Patnaik, Sidhartha Das & B.K. Patniak[33]

Current knowledge reveals that HGU is usually normal in Type-2 diabetics while HGO is high due to hepatic insulin resistance [lead to repression of key hepatic glycolytic enzymes and depression of glucogenic enzymes] and less of futile cycles of carbohydrate metabolism taking place with in the hepatocytes[34,35]. Leading to decreased trapping of insulin by liver causing excess liberation of insulin into systemic circulation leading to hyper insulinemia[36].

LB Type-2 diabetics have moderately severe to severe basal hyperglycemia. According to Das there was a negative correlation between FBG and BMI which negated the likely possibility of insulin resistance in the peripheral tissue and hence glucose handling of the liver by LB type diabetics demands special attention. But low circulating levels of insulin is a universal observation[37]. This indicate

that hyperactive metabolic state observed in the liver of these diabetics having low body weight is probably an inherent characteristic in them which is responsible for excess utilization of insulin during it first pass. There is probably excess extraction of insulin in the portohepatic circulation leading to lower levels of circulating insulin. This may be due to excessive futile cycle of carbohydrate metabolism in the liver.

The LB Type-2 diabetics had the highest FBG vis a vis highest circulating levels of glucokinase suggests excess of futile cycles of carbohydrate metabolism in liver especially inter conversion of glucose to glucose phosphate and again back to glucose. The reconverted glucose is liberated into the circulation causing excess rise of hepatic glucose output.

8.UK PDS[38,39]

In UKPDS cohort of 3672 patient 10% newly diagnosed NIDDM patient were positive for ICA and 6% for GADA. The frequency of ICA and GADA were higher in those with a younger age at onset. Most importantly ICA or GADA positive patient require insulin earlier and more frequently than patient negative for antibodies. In UKPDS report among patient less than 35 years of age at onset with GADA or ICA positively lead to an insulin requirement in 84% and 94% of NIDDM individuals by 6 yrs. Thus GADA may be a better predictor of insulin requirement than other clinical (low BMI) or biochemical (C-peptide levels) parameters.

9.Tuomi et al[40,41]

On the basis of study in Finland reported that 9.3% of the Type-2 DM patients were positive for GADA. In contrast to GADA the frequency of IA-2 antibodies directed against the antigen tyrosin phosphates are in frequent in Type-2 diabetics. They detected IA-2 antibodies in only 17% of GADA positive individuals and in 0.5% of those in whom GADA was absent.

10. Kanungo. A, Shtauvene. A, Samak.K.C. et al[42]

They evaluated 72 North Indian Type-2 DM patient and reported a prevalence of 25% for GADA, which was similar to prevalence in Type-1 DM patient. While only 4% of Type-2 diabetes mellitus were tyrosine phosphatase (IA-2) positive Vs 22% of Type-1 DM. Thus IA-2 antibodies are likely to be better discriminator of Type-1 & Type-2 DM.

In visible absence of ICA in low body weight Type-2 diabetics with fairly preserved C-peptide level and reserve suggest that they are immunologically classical Type-2 diabetics and not slowly evolving Type-1 DM[43].

11. Irivin et al[44,45]

According to them GADA may be a better predictor of insulin requirement in Type-2 DM patient than other clinical low BMI or biochemical C-peptide level parameters. Patients having secondary failure to OHA were likely to be positive for GADA or ICA[46].

12. Siddartha Das and Baruna Mishra[48]

They took 146 newly diagnosed and untreated cases of Type-2 DM without any other major illness and were recruited for the evaluation of dietary habit and its impact on metabolic profile.

Detailed dietary counseling was done. The LB were asked to increase intake by more than 350 Kcal/day while obese were asked to cut down their intake by 900 Kcal/day. During follow up marginal improvement in mean BMI was observed in low body weight subjects.

From the dietary and nutrition point of view the best balanced were the standard weight subject. The alteration and influences of dietary components in the over weight diabetics was similar to most of the international data with negative influence of carbohydrate diet on blood glucose. In LB Type-2 group an inherent defect for carbohydrate handling was obvious. Both over weight and low body weight Type-2 diabetics probably represent the two end of the U shaped curve with regards to the metabolism and blood glucose levels[49].

13. S. Banerjee and U.K. Pal[11,32,47].

They selected 75 cases of Type-2 DM (25 cases of lean 25 cases of non-obese and 25 cases of obese) after exclusion hepatic endocrine cardiorespiratory and other systemic diseases.

This study revealed a male predominance (3:2) amongst LB Type-2 DM. The mean age of presentation was in mid forties and most were presented with usual symptoms. The prevalence of family history was much higher. These LB Type-2 DM were not restricted to a poor socioeconomic status but were more from rural areas as compared to non obese and obese subject FBG in the LB Type-2 was not statistically different from either non obese or obese Type-2. The fasting C-peptide levels were lower than those in other two groups of diabetics, which suggest that level of fasting hyper insulinemia. But the rise in the plasma level of C-peptide was much

more brisk than the latter group, following diet intake near normal C-peptide levels vis a vis basal hyperglycemia is an important characteristic of LB Type-2 diabetics. Probably serve as a marker for the low circulating levels of insulin with fairly preserved β cell reserve. Therapeutically a large number of LB subjects respond well to oral hyper glycemic agent.

14. G.R.Sridhar, S.Veena, K.Madhu[50,51]

In this study characterized the clinical profile of a group of LB Type-2 and evaluated the quality of life well being and psychological adjustment to diabetes. They conducted similar clinical profile to other study but LB Type-2 DM had a higher prevalence of sleep disturbance than other group. Finally they scored lower on quality of life well being psychological adjustment to diabetes scales compared to non-lean Type-2 diabetics. Thus the common phenotype runs through in patients with LB Type-2 DM who have a poor quality of life than their peer's. They therefore need appropriate evaluation and extra attention for better management.

15. Siddartha Das,R.K.Mishra,B.B.Jena, B.K. Mishra, K.C.Mishra, and B.Sarangi[52]

In their study they observed that mortality amongst hospitalized patient with non insulin dependent diabetics mellitus (NIDDM) was 20%. Major causes of mortality are cardio vascular accident, Ischemic heart diseases & infections.

16. Siddartha Das, E.A.Sotaniemi, A.K. Baliarsinha and A,Rautia[53,54,55].

They interpreted that, the LB Type-2 diabetics seen in India not only have hyperactive hepatic metabolic state but such metabolic

process is inherent to them and not influenced by other co-existence factors. The hyperactive hepatic metabolic state is very likely responsible for excess extraction / trapping of insulin during its first pass which consequently leads to low circulating insulin levels. This reasonably explains the insulin-C peptide disparity observed in LB Type-2 diabetics. Such metabolic activities causes excess of futile cycles which not only dissipate energy, utilize insulin but causes raised FBG levels.

17. B.C.Patnaik, S.R.Patnaik, A.C.Mahakur, U.C.Tripathy, H.Sahu, S.C.Mishra[56,57,58,59]

Glycosylated haemoglobin (HbA_I) is formed by the addition of a molecule of glucose to the N-terminal valine of one or both beta chains of adult haemoglobin by a spontaneous and non-enzymatic reaction. Apart from diabetes, HbA_I is altered in many physiological and pathological states like pregnancy, iron deficiency anaemia, haemochromatosis and renal failure.

In renal failure, shortened life span of erythrocytes is well known and low levels of HbA_I are expected[1]. On the other hand a raised HbA_I is possible because there occurs a disordered carbohydrate metabolism in renal failure. While a majority of workers have noted increased levels of HbA_I in chronic renal failure (CRF), the rise being more pronounced in diabetic nephropathy, some demonstrated decreased levels.

They concluded that glycosylation of haemoglobin is influenced by glycemic status as well as shortened life span of erythrocytes in renal failure. Thus decreased HbA_I is possible in both diabetic and non-diabetic renal failure. One must be cautious in interpreting the

glycosylated haemoglobin values as an indicator of diabetic long term control in the presence of renal failure.

18. K.C.Samal, Sidhartha Das, C.R.Parija, B.B.Tripathy[60,61,62]

They concluded that C-peptide has no biological activity. In the serum of normal subjects C-peptide is much higher than proinsulin and its intermediates. Proinsulin reacts less well than C-peptide with C-peptide antiserum. Direct assay of total serum CPR in normal subjects represents mainly C-peptide level. Hence it is justified to presume that the value of CPR is very close to C-peptide level, particularly in insulin deficient diabetics in whom the proinsulin level in circulation is negligible. Further we selected patients who were not on insulin therapy, thus excluding the possibility of any interference by exogenous proinsulin.

As anticipated, plasma levels of glucose were remarkably higher following oral glucose than mixed meal and values of IRI both basal as well as in response to glycemic stimuli, were low in both groups of patients. Inspite of this there was no significant difference in the IRI level following glucose are mixed meal in either group of patients suggesting poor insulinogenic reserve. The mean CPR level observed in the present study are lower than those reported from the west, both at basal and one hour after oral glucose. This is consistent with our previous observation of low insulin level in the local population. Result of this study indicates that residual B cell function, although adequate to maintain near normal basal CPR level in freshly detected case of FCPD, appear to be grossly insufficient to raise hormone production appropriately in response to glycemic stimuli. It

was further observed that although the difference between the rise of IRI following mixed meal and oral glucose was not statistically significant in normal controls the difference in the corresponding CPR values was highly significant ($P < 0.01$). This suggests that CPR is a better indicator of B cell performance than plasma insulin, passively due to insignificant trapping of C-peptide level in the liver. In freshly detected case of IDDM on the other hand lower level of CPR suggest that B cell function compromised even in the basal state. Yet, definite rise in C-peptide value, following both oral glucose and mixed meal indicate presence of certain quantum of residual β cell activity. It is well known that most of these patients ultimately develop complete loss of β cell function in course of time. The remarkably lower incidence of ketosis in FCPD as compared to IDDM may be an account of better residual β cell function as indicated by higher CPR level at basal state and in response to glucose challenge. However, the relatively lower difference in the peripheral IRI levels of these two groups of diabetics can be explained by the well known fact that a larger proportion of insulin (70-90%) is trapped by the liver in diabetics.

Aims

&

Objectives

Aims and Objectives

1. To study the clinical and biochemical profile of low body weight Type-2 diabetes mellitus.

2. Do these patient differ clinically and biochemically from other Type-2 diabetes mellitus patients?

Material & Methods

Material and Methods

The present study was carried out in the Department of Medicine M.L.B. Medical College Jhansi in 2001 - 02. In this study 30 patients of Type-2 DM whose BMI <18.5 (low body weight) were enrolled for study group, 25 normal body weight (BMI between 18.5 - 25) and 20 were over body weight (BMI >25).

Selection of cases

For this study patients were selected from

1. General Medicine OPD
2. Diabetic Clinic OPD
3. Gynaecology & Obstetrics OPD
4. Skin OPD
5. Tuberculosis & Chest OPD
6. Patient admitted in Ward

Criteria for selection

Any individual who volunteered himself or herself fulfilling the criteria either of LB Type-2 DM i.e., BMI <18.5% kg/mt^2 or non-obese 18.5 – 25 or obese > 25. The Type-1 DM patients were excluded on the basis of age, clinical presentation and insulin dependence from starting.

Method of study

Study of clinical profile includes following :-

1. Full clinical history about disease and it complication & total duration of illness.
2. Family history maternal or paternal and siblings.
3. Age and sex of the patient.
4. Educational status.
5. Dwelling – Rural or Urban.
6. Socioeconomic status of the patient dividing into
    i) Lower class
    ii) Middle class
    iii) Upper class
7. Dietary history
8. Habit

    Vegetarian / Non Vegetarian
    Alcoholic / Non Alcoholic
    Smoker / Non Smoker
    Tobacco Chewer / Non Tobacco Chewer
    Other
9. Occupational history
10. General Examination includes
    - Built and body proportion
    - Nutrition
    - Pallor, Icterus, Cyanosis, Clubbing
    - Edema, Lymphadenopathy
    - Skin, hair, Nails

- Vertebral column and joint
- Temperature
- Pulse
- Blood pressure
- Respiration

11. Systemic Examination

❖ CNS

- Higher functions
- C.N.
- Motor system
- Sensory system
- Other

❖ CVS

❖ Respiratory

❖ Abdomen

❖ Others

12. Investigation

❖ Haemogram

❖ Diabetic profile including :-

- Fasting blood sugar
- HbA_1c
- Anti insulin antibodies
- Insulin
- Anti microsomal antibodies
- C-peptide.

- ❖ Renal parameters
  - Urine – Routine and microscopic examination
  - 24 hours urinary protein
  - Serum creatinine
- ❖ Lipid profile
  - Total serum cholesterol
  - High density lipoprotein
  - Low density lipoprotein
  - Very low density lipoprotein
- ❖ X-ray Chest PA view
- ❖ Fundus
- ❖ ECG
- ❖ Echo

Specimen collection and handling

A.Blood collection

Collect 10 ml of fasting blood sample from venipuncture and dispense it as mentioned below.

1. Double OX tube 2ml blood for haemogram.
2. Fluoride tube 2ml blood for fasting blood sugar
3. EDTA tube 2 ml blood for HbA_1c.
4. Plain tube AMD insulin, C-peptide, Serum Creatinine 4ml blood.

B.Urine collection

Collect 2 ml of urine from 24 hrs urine pool in plain tube and note the total 24 hrs urinary volume for micro albuminurea.

C. Samples can be stored at 2-8^{0}C upto 24 hours, for longer periods store samples at –20^{0}C. Avoid repeated freezing and thawing.

Methods used

Haemogram – Biochemistry Dox blood

Fasting blood sugar – Biochemistry Plasma

HbA$_I$c – High performance liquid chromatography

AIA – Radio immuno assay

Insulin – Chemiluminescence Immuno assay

AMA – Haemoglutination assay

C- peptide - Chemiluminescence Immuno assay

Urine – Biochemistry and Chemiluminescence Immuno assay

S.Creatinine – Biochemistry

Lipid profile – Biochemistry

These Investigations were conducted at the Thyrocare laboratory.

HbA$_I$C (GLYCATED HEMOGLOBIN)

The other important advancement in diabetes management is the glycosylated hemoglobin assay. In normoglycemic subjects a small proportion of hemoglobin A is attached to a carbohydrate moiety, thus creating what is called glycosylated or glycated hemoglobin. The glycosylated hemoglobin can be separated into three distinct fractions, which are designated A_{1a}, A_{1b}, and A_{1c}. Because of electrophoretic behaviour of these minor hemoglobins, they are referred to as fast hemoglobin.

In conditions of sustained hyperglycemia, such as in diabetes mellitus, the proportion of hemoglobin that is glycosylated is increased substantially. This glycosylation is the result of post-translational modification of hemoglobin A molecules; the binding of glucose is a non-enzymatic process that occurs continuously during the life of the red blood cell. Thus, the amount of glycosylated hemoglobin reflects the glycemic control of a patient during the 6 to 8 week period before the blood sample was obtained.

Glycosylated hemoglobin can be measured by chromatographic, chemically based, or electrophoretic assays. The advantages and disadvantages of these assays will be reviewed briefly. Falsely high levels of glycohemoglobin can be produced in chromatographic assays by carbamylated hemoglobin (formed in uremia), acetaldehyde addition (in alcoholics), or increased fetal hemoglobin (elevated in thalassemia, aplastic anemia, myeloproliferative disorders, and pregnancy). Hemoglobin variants such as HbS or HbC can cause falsely low results in these assays.

The labile fraction of glycohemoglobin (reflects reversibly bound component) is measured in ion-exchange assays but not in chemical assays employing affinity chromatography or calorimetry. Pretreatment of the sample in ion-exchange assays overcomes this problem.

The electrophoretic method that uses agar gel electrophoresis measures all of HbA_1, whereas isoelectric focusing on polyacrylamide gels gives wider separation of the different hemoglobin components and therefore can quantitate HbA_{1c}. In fact, while affinity chromatography measures all glycosylated hemoglobin, it is not

affected by temperature or by the presence of hemoglobin variants or fetal hemoglobin. In general, ion-exchange columns (measure Hb_{A1}) correlate quite well with affinity columns (measures all glycosylated hemoglobins) even though they measure two different substances. The glycosylated hemoglobin test gives an estimation of the average glycemic level during the 6 to 8 weeks preceding the test. It correlates well with fasting and postprandial blood glucose levels and 24-hour urinary glucose levels. The glycosylated hemoglobin assay is presently one of the most widely applied tests in the management of diabetes. It is useful for the assessment of glycemic control in both patients with Type-I and Type-II diabetes.

Glycosylated hemoglobin values must be assessed with caution in patients with unstable diabetes. Levels of blood sugar in these patients fluctuate from very low to very high on an almost daily basis, a situation that can lead to unwanted symptoms of hyperglycemia and dangerous episodes of hypoglycemia. The assay of glycosylated hemoglobin should be done every 3 to 4 months, with the goal of adjusting therapy to obtain the lowest value that does not place patients at undue risk for hypoglycemic reactions.

HbA_1c values

< 6%	Normal
6 – 7	Good Control
7 - 9	Avg. Control
>9	Poor Control

C-Peptide assays

The pancreatic β-cell's primary function is the production, storage and regulated secretion of insulin. Under normal circumstances, the β-cell maintains a condition where there is always readily available pool of insulin that can be rapidly secreted in response to a stimulus, such as a rise of blood glucose. Any increase in insulin release is compensated for by a corresponding increase in insulin biosynthesis, so that β-cell insulin stores are constantly maintained.

Insulin is produced in the β-cells of the islets of Langerhans by cleavage of its precursors, proinsulin, into one molecule of insulin and one molecule of C-peptide. Insulin is subsequently released into the circulation at concentrations equimolar to those of C-peptide. Small amounts of intact proinsulin and proinsulin conversion intermediates are also released. Their low concentration in serum, however, ensures that under normal physiological conditions, their in vivo effects are negligible. In subjects with non-insulin-dependent diabetes mellitus (NIDDM) and even impaired glucose tolerance, however, there is a disproportionate increase in proinsulin-related peptides to approximately 30% of the total immunoreactive insulin in serum.

In contrast to insulin and proinsulin, C-peptide does not appear to be metabolically active. It is considered to be a good marker of insulin secretion because of its equimolar secretion with insulin, negligible hepatic extraction, and constant peripheral clearance at different plasma concentrations and in the presence of alterations in plasma glucose concentrations. The kidney exclusively excretes it,

and its plasma half-life is approximately 30 minutes contrast sharply with the short plasma half-life (approximately 4 minutes) of insulin.

Insulin secretion rates cannot be calculated directly from the peripheral insulin concentrations, because insulin secreted into the portal vein by the pancreas is taken up to a large and variable degree by the liver before it enters the general circulation. Measurements of plasma insulin concentrations are further complicated in insulin-treated patients because of the presence of circulating insulin antibodies, and because insulin immunoassays cannot distinguish endogenous from exogenous insulin. Therefore, peripheral C-peptide concentrations reflect the secretion of the β-cells more accurately than insulin. For these reasons, measurements of C-peptide in plasma have entered general use as a measure of β-cell function.

Clinical applications of C-peptide

1. To assess the residual β-cell function in patients with insulin and to distinguish between IDDM and NIDDM. Of particular interest is its use to indicate the need for progression to insulin therapy in NIDDM.
2. The diagnosis of factitious hypoglycemia. The surreptitious administration of insulin causes high insulin levels in absence of elevated C-peptide concentrations.
3. To diagnose the presence of insulinoma.
4. To assess residual pancreatic tissue after pancreatectomy.

Insulin Assays

Insulin assays play a central role in the investigation of glucose metabolism disorders. Particularly useful in the investigation of the causes of hypoglycemia, insulin assays are also used in the determination of the pathogenesis of Type-I and Type-II diabetes, assessment of β-cell function and for studies on the pharmacology of insulin itself.

Insulin is a polypeptide consisting of two chains linked by disulphide bonds. The A and B chains consist of 21 and 30 amino acids respectively, with disulphide bonds located at positions A7-B7 and A20-B19. The A chain also has an internal disulphide bond bridging the amino acids A6 and A11. Insulin and C-peptide are secreted in equimolar amounts into the portal circulation together with small quantities of proinsulins. 2-6% of the insulin released from the secretory granules is actually in the form of proinsulins and these represent 5-10% of the bioactivity of insulin.

In human circulation, the half-life of insulin is approximately 3-5 minutes. Almost all tissues have the ability to metabolize insulin, but 80% is degraded in the liver and kidneys. In fact 50% of insulins is removed in a single pass through the liver. As the liver does not remove proinsulins and C-peptide, they accumulate in blood and account for 15-20% of the total amount of insulin and proinsulins in the basal state. Glucose is the primary signal that stimulates insulin secretion. Therefore, to correctly interpret an insulin measurement, a simultaneous measurement of the glucose level is also needed.

Clinical uses of insulin measurements

The main clinical application of plasma insulin measurements is in the investigations of the causes of hypoglycemia. This disorder can be caused by hyperinsulinism, insulinoma, insulin autoimmune syndrome or by non-insulin mediated factors. For epidemiological use, insulin assays have been proposed as a marker or risk factor for the development of coronary heart disease and as an early marker for the development of diabetes.

Impaired glucose tolerance (IGT) and diabetes mellitus are diagnosed solely on the basis of chronic hyperglycemia. However, insulin measurements are used in research to study the pathogenesis of these disorders. In Type-1 diabetes, insulin measurements can be useful in pharmacological studies. Type-2 diabetes results from insulin resistance associated with an insulin secretory defect. Specific insulin assays are used to determine the relative insulin deficiency, and the inability of the pancreas to compensate for insulin resistance by adequate insulin secretion. Insulin measurements have a clinical value in the diagnosis of severe insulin resistance. Combined with C-peptide determination, insulin measurements may be used to assess the residual β-cell function, especially in newly diagnosed cases of Type-1 diabetes. They may also aid in the discrimination between Type-1 and Type-2 diabetes.

The routine performance of an insulin assay may be hampered by factors related to the sample material; insulin auto-antibodies are frequently present in prediabetic states as well as in recent onset IDDM and other antibodies with equally disturbing capabilities have

been reported to be frequently present also in the general population. Heterologous antibodies and rheumatoid factors are also known to be potentially disturbing factors. The influence of such antibodies may be minimized by precipitation with polyethylene-glycol at the time of sampling, followed by the determination of free insulin immuno-reactivity in the supernatant. Haemolysis is another sample derived factor which influences most insulin measurements due to the presence of insulin degrading enzymes in erythrocytes. The impact of these types of interference on the individual insulin assay is only weakly predictable and must therefore be studied experimentally. An example is the influence of haemolysis: in competitive assays, where an insulin antibody is used in limited concentration, released enzymes may lead to insulin degradation into large fragments (half-molecules) each of which are still capable of reacting in the assay and with similar efficiency as insulin itself. In two – site assays the simultaneous presence of two immunochemical sites on the analyte-molecule are needed to allow determination. Insulin fragments may not be reactive at all. Thus, the analytical design selected has an influence on the impact of a potentially interfering factor.

Anti Insulin Antibodies

Anti insulin antibodies interfere in insulin RIAs and IMAs. They are present in the serum of patients with insulin autoimmune syndrome and are frequently found in the serum of insulin – treated diabetic patients, even when the patients receive biosynthetic human insulin. Studies have indicated that 30% of Type-1 diabetic patients have anti-insulin antibodies prior to insulin administration, and 50% of

diabetic patients treated with human insulin have these antibodies. In the serum of these patients, insulin circulates in both the bound form and also as the free, unbound and biologically active moiety. In RIAs the size of the effect of anti insulin antibody interference depends mainly on the technique used to separate free and bound radioligand and can thus yield either falsely – elevated or falsely – low insulin values. In two site immunoassays, if the affinity of the antibodies used in the assay exceeds that of the anti insulin autoantibodies, falsely high free insulin values may be produced. On the other hand, if the assay antibodies are less avid than the autoantibodies, the final result will tend to reflect the free insulin concentration, since little or no displacement of the autoantibody-insulin complex occurs during the incubation time.

Anti Microsomal Antibody (AMA or anti- TPO antibodies)

Anti Microsomal Antibody are those that are directed against the microsomes in the thyroid gland. Abnormal values are seen in Hashimoto's thyroiditis (100%), Graves disease (80%), hypothyroidism and atropic thyroiditis. Estimation of AMA would help the physician in planning a long term treatment strategy for a patient of thyroid dysfunction.

Serum creatinine

Creatinine, an end product of creatine, which is excreted by kidneys and it is used to monitor renal diseases. In patients with renal dysfunction, the serum creatinine is disturbed resulting in accumula--tion of creatinine in serum. In diabetics, high serum creatinine is of bad prognostic value and signals deterioration of renal function.

Urinary Albumin / Urinary Creatinine Ratio

Whenever urinary concentration of an analyte is to be co-related, it is essential to know what volume of urine output in 24 hours is collected. However, in certain emergencies or in a situation, where collection of 24 hours urine is not practical, estimation of urinary analytes is done on random samples. It is expected that urinary creatinine levels (which co-relates to urine volume) would be indicating the role of urinary volume changes in albumin concentration and therefore in such circumstances albumin to creatinine ratio is demanded for any meaningful clinical co-relation.

Normal Albumen	< 30 μg/mg creatinine
Micro Albumen	30 – 300 μg/mg creatinine
Macro Albumen	> 300 μg/mg creatinine

Micro Urinary Albumin

Diabetic patients are prone to develop kidney damage as the disease progresses. The damage caused to the kidney if known in time can be reduced or eliminated by giving the patient certain drugs and full blown diabetic kidney damage can be avoided. Detection of increased levels of albumin in the urine at an early stage serve this purpose as it is the earliest marker of diabetic marker of diabetic kidney disease. It may be worth noting that various other disorders also result in nephropathy (kidney disorders).

Normal Albumin Urea	< 20 μg/min
Micro Albumin Urea	20-200 μg/min
Macro Albumin Urea	>200 μg/min

Working Proforma

1. Case No: MRD/OPD No: Date: / /
2, Patient Name:
3. Address: Tel:
4. Date of Birth:
5. Date of follow up
6. Age: Sex: M / F Occupation:
7. Residence: Rural / Urban Literacy status:
8. Socio Economic status: Lower / Upper / Middle
9. OPD Attendance: Regular / Irregular
10. Total Duration of illness: Age at Diagnosis
11. Complaints at Diagnosis:
12. Present Complaints:
13. Past History:
14. Family History and Relation: Relation:
15. Habit
    - Vegetarian/Non-Vegetarian
    - Alcoholic/Non-Alcoholic
    - Smokers/Non-Smokers
    - Tobacco Chewer
    - NonTobacco Chewer
    - Others
16. Associate Illness

Illness	Present / Absent	Duration
Hypoglycemia		
Hyperglycemia		
Ketosis		
CVD		
PVD		

CVA

Cataract

Retinopathy

Nephropathy

Peripheral Neuropathy

Sensory Neuropathy

Motor Neuropathy

Mixed Neuropathy

Autonomic Neuropathy

Impotency

GI Symptoms

Diabetic Foot

Tuberculosis

Skin Infection

Fungal Infection

Other

17. General Examination Pulse rate /min

Blood Pressure

Supine position mm/Hg

Standing (After 3 min) mm/Hg

18. Anthropometry Weight Kg Height mt. BMI

Waist cm Hip cm W/H

19. Systemic Examination:

CNS

Higher Function Cranial Nerves

Motor System Sensory System

Others

CVS

Respiratory System

Abdomen

Other

20. E.C.G
21. ECHO
22. X-ray
23. Fundus
24. Biochemical Result
    1. F.B.G (mg %)
    2. HbA_1c (%)
    3. Anti Insulin Antibodies (%)
    4. Insulin (μ I.U./ml)
    5. Antimicrosomal Antibodies (Titer)
    6. C-Peptide (ng / ml)
    7. Urine Albumin (μg)
    8. S.Creatinine (mg)
    9. Urine Albumin / Creatinine (μg/mg)
    10. Lipid Profile

        T.Ch. (mg %) HDL (mg %)

        LDL (mg %) VLDL (mg %)

        S.Triglyceride (mg %)

        Tc/HDL LDL / HDL
25. Treatment
26. Treatment Complains
27. Diabetic Control Poor / Average / Good

Observations

Observations

The present study was carried out in the Department of Medicine M.L.B. Medical College Jhansi In 2001 - 02. In this study 30 patient of Type-2 DM whose BMI <18.5 (low body weight) were enrolled for study group and Type-1 DM patient were excluded on the bases of age of onset, clinical presentation and insulin dependency from starting. Because Type-2 DM usually occurs in normal body weight (BMI between 18.5 -25) and over body weight (BMI >25) therefore they were taken as control I (BMI 18.5 – 25) 25 patient and control II (BMI >25) 20 patient.

Their complete history, clinical examination and investigation done. General characteristics of the patients were as follows:-

Table 1. Distribution of patients according to their BMI in study group (group A), control group I (group B) and control group II (group C)

Subject	Group A	Group B	Group C
Type of patient	Study Group	Control I	Control II
Total No of patients	30	25	20
BMI Range	< 18.5 (LB Type-2)	18.5 – 25 (Normal body weight)	> 25 (Over body weight)
BMI mean ± SD	17.19 ± 1.03	22.56 ± 1.54	28.9 ± 3.91

This table shows that in this study patients are placed in three groups. Group A called study group, BMI < 18.5 (mean ± SD 17.19 ± 1.03) total No. of patient 30, Group B called control I, BMI 18.5 – 25 (mean ± SD 22.56 ± 1.54) total No. of patient 25 and Group C called control II, BMI >25 (mean ± SD 28.9 ± 3.91) total no. of patients 20.

Table 2. Distribution of patient according to their sex in study group (group A), control group I (group B) and control group II (group C)

Sex	Group A (n=30)		Group B (n=25)		**Group C** (n=20)	
	No.	%	No.	%	No.	%
Male	22	72.6	20	80	14	70
Female	8	27.4	5	20	6	30

Statistical significance calculated :-

Between A and B groups

df = 53

t value = 0.61

p value = > 0.5

Between A and C groups

df = 48

t value = 0.20

p value = > 0.8

This table shows that in Group A Male 22(72.6%), Female 8(27.4%) out of 30. Group B Male 20(80%) Female 5(20%) out of 25. Group C Male 14(70%) Female 6(30%) out of 20. Their was no statistical significant difference between A and B group (p > 0.5) and A and C group (p > 0.8). Thus sex distribution is almost similar in these groups.

Table 3. Distribution of patient according to their age of onset of diabetes in study group (group A), control group I (group B) and control group II (group C)

Age	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
≤ 40	7	23.3	4	16	7	25
41 – 60	21	70	20	80	11	65
≥ 61	2	6.7	1	4	2	10
Mean ± SD	47.7 ±	8.8	47 ±	7.4	44.75 ±	9.3

Statistical significance calculated :-

Between A and B groups

df = 53

t value = 0.93

p value = > 0.9

Between A and C groups

df = 48

t value = 3.50

p value = < 0.001

This table shows that in group A ≤ 40 yrs. 7(23.3%), 41 – 60 yrs. 21(70%) ≥ 61yrs. 2(6.7%) and mean ± SD age 47.7 ± 8.8. Group B ≤ 40 yrs 4(16%) 41 – 60 yrs 20(80%). ≥ 61yrs 1(4%) and mean ± SD 47 ± 7.4 while Group C ≤ 40 yrs 7(25%) 41 – 60yrs 11(65%) ≥ 61yrs 2(10%) mean ± SD 44.75 ± 9.3. There was no statistical significant difference between age A and B group (p >0.9), while group C are younger according to age of onset and significant difference between A and C group (p < 0.001).

Table 4. Distribution of patient according to total duration of diabetes in study group (group A), control group I (group B) and control group II (group C)

Total Duration of Diabetes (yrs)	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
≤ 1	2	6.7	4	16	3	15
2 – 5	18	80	17	68	10	50
6 – 10	7	23.3	3	12	5	25
≥ 11	3	10	1	4	2	10
Mean ± SD	4.8 ±	3.14	3.8 ±	2.57	4.5 ±	3.14

Statistical significance calculated :-

Between A and B groups

df = 53

t value = 2.2

p value = < 0.05

Between A and C groups

df = 48

t value = 0.6

p value = > 0.5

This table shows that in group A total duration ≤ 1 yr. 2(6.7%), 2 – 5 yrs. 18(60%) 6 – 10 yrs 7(23.3%) ≥ 11yrs 3(10%) and mean ± SD 4.8 ± 3.14. Group B ≤ 1 yr. 4(16%), 2 – 5 yrs. 17(68%) 6 – 10 yrs 3(12%) ≥ 11yrs 1(4%) and mean ± SD 3.8 ± 2.57.while Group C ≤ 1 yr. 3(15%), 2 – 5 yrs. 10(50%) 6 – 10 yrs 5(25%) ≥ 11yrs 2(10%) and mean ± SD 4.5 ± 3.14.

There was statistical significant difference according to total duration of diabetes between A and B group ($p < 0.05$) and no statistical difference between A and C ($p > 0.5$) total duration of diabetes wise.

Table 5. Distribution of patient according to their residence in study group (group A), control group I (group B) and control group II (group C)

Residence	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
Rural	15	50	10	40	8	40
Urban	15	50	15	60	12	60

Statistical significance calculated :-

Between A and B groups

df = 53

t value = 0.76

p value = > 0.4

Between A and C groups

df = 48

t value = 0.77

p value = > 0.4

This table shows that in group A patient of rural residence 15(50%) and urban 15(50%). Group B rural 10(40%) and urban 15(60%) while in group C rural 8(40%) and urban 12(60%). Thus there was no statistical significant difference between A and B group ($p > 0.4$), and A and C ($p > 0.4$) residence wise.

Table 6. Distribution of patient according to their socioeconomic status in study group (group A), control group I (group B) and control group II (group C)

Residence	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
Lower	7	23.3	8	32	2	10
Middle	20	66.6	14	56	14	70
Upper	3	10	3	12	4	20

This table shows that in group A lower class 7(23.3%) middle 20(66.6%) upper 3(10%), group B, lower 8(32%), middle 14(56%), upper 3(12%), while group C lower 2(10%) middle 14(70%) upper 4(20%).

Statistical significance calculated: -

Class	Between A and B group	Between A and C group
	df 53	df 48
Lower Class	t value 0.73	t value 1.22
	p value > 0.4	p value > 0.2
Middle Class	t value 0.82	t value 0.25
	p value > 0.4	p value > 0.8
Upper Class	t value 0.24	t value 1.03
	p value > 0.8	p value > 0.3

Thus this table shows that there was no statistical significant difference in socioeconomic status between A and B, and A and C and most of the patients were of middle class and upper class.

Table 7. Distribution of patient according to their literacy status in study group (group A), control group I (group B) and control group II (group C)

Literacy status	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
Literate	20	66.7	17	68	17	85
Illiterate	10	33.3	8	32	3	15

Statistical significance calculated :-

Between A and B groups

df = 53

t value = 0.10

p value = > 0.9

Between A and C groups

df = 48

t value = 1.47

p value = > 0.1

This table shows that in group A literate 20(66.7%) Illiterate 10(33.3%) group B literate 17(68%). Illiterate 8(32%) and group C literate 17(85%) Illiterate 3(15%).

Thus there was no statistical significant difference in status between A and B ($p > 0.9$) and A and C ($p > 0.1$).

Table 8. Distribution of patient according to their occupation in study group (group A), control group I (group B) and control group II (group C)

Occupation	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
Housewife	8	26.6	5	20	6	30
Business	6	20	3	12	4	20
Farmer	10	33.4	7	28	5	25
Service	6	20	10	40	5	25

This table shows that in group A 8(26.6%) housewife, 6(20%) business, 10(33.4%) farmer, 6(20%) service class. Group B 5(20%) housewife, 3(12%) business, 7(28%) farmer, 10(40%) service class while in Group C 6(30%) housewife, 4(20%) business, 5(25%) farmer, 5(25%) service class.

Table 9. Distribution of patient according to their habits in study group (group A), control group I (group B) and control group II (group C)

Habit	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
Non-vegetarian	14	46.7	13	52	10	50
Alcoholic	11	36.7	9	36	6	30
Smoker	13	43.3	10	40	7	35
Tobacco chewer	14	46.7	13	52	8	40

This table shows that in group A 14(46.7%) Non-vegetarians, 11(36.7%) Alcoholic, 13(43.3%) Smoker, 14(46.7%) Tobacco chewer. Group B 13(52%) Non-vegetarians, 9(36%) Alcoholic, 10(40%) Smoker, 13(52%) Tobacco chewer while in Group C 10(50%) Non-vegetarians, 6(30%) Alcoholic, 7(35%) Smoker, 8(40%) Tobacco Chewer.

Table 10. Distribution of patient according to their family history of diabetes in the study group (group A), control group I (group B) and control group II (group C)

Family History	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
Mother	1	3.3	1	4	0	0
Father	2	6.6	1	4	1	5
Sibling	6	20	1	4	0	0
Total	9	30	3	12	1	5

This table shows that in group A family history of diabetes in mother 1(3.3%), father 2(6.6%), sibling 6(20%), total 9(30%). Group B in mother 1(4%), father 1(4%), sibling 1(4%), total 3(12%) while in group C in mother 0, father 1(5%), sibling 0, total 1(5%).

Statistical significance calculated: -

	Between A and B group	Between A and C group
	df 53	df 48
Mother	t value 0.14 p value > 0.8	t value 0.30 p value > 0.7
Father	t value 0.13 p value > 0.6	t value 1.19 p value > 0.2
Sibling	t value 1.91 p value < 0.05	t value 2.16 p value < 0.05
Total	t value 1.94 p value < 0.05	t value 2.20 p value < 0.05

There is no statistical significance in mother and father family history in between A and B group, A and C group while significant difference in between sibling that in higher in group A(p value in between A and B = < 0.05 and A and C = < 0.05), total family history also higher in group A(p value A and B = < 0.05 and A and C = < 0.05).

Table 11. Distribution of patient according to complain at diagnosis in the study group (group A), control group I (group B) and control group II (group C)

Complain	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
Classic	21	70	24	96	19	95
Parasthesia and Numbness	11	36.7	4	16	1	5
Weakness	15	50	4	16	2	10
DF	2	6.6	1	4	0	0
Fever and cough	4	13.2	1	4	0	0
Other	1	3.3	0	0	1	5

Statistical significance calculated: -

	Between A and B group	Between A and C group
	df 53	df 48
Classic Complain Parasthesia and numbness Weakness	t = 2.5 p < 0.02 t = 1.69 p < 0.05 t = 3.0 p < 0.01	t = 2.2 p < 0.05 t = 2.6 p < 0.02 t = 3.3 p < 0.01

This table shows that classic complain at diagnosis is statistically significant difference between A and B group ($p < 0.02$) and A and C group ($p < 0.05$) that is lesser in group A, while parasthesia numbness and weakness higher in group A with statistically significant difference between A and B, A and group C.

Table 12. Distribution of patient according to their waist in the study group (group A), control group I (group B) and control group II (group C)

Waist in cm.	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
≤ 70	13	43.3	0	0	0	0
71 – 80	17	56.7	0	0	0	0
81 – 90	0	0	14	56	8	40
91 – 100	0	0	11	44	1	5
≥ 101	0	0	0	0	11	55
Mean ± SD	71.7 ± 3.8		89.6 ± 4.2		100.2 ±13.3	

Statistical significance calculated :-

Between A and B groups

df = 53

t value = 33.47

p value = < 0.001

Between A and C groups

df = 48

t value = 37

p value = < 0.001

This table shows that in group A waist size ≤ 70 cm 13(43.3%), 71 – 80 cm 17(56.7%) Mean ± SD 71.7 ± 3.8. In group B 81 – 90 cm 14(56%), 91 – 100 cm 11(44%) Mean ± SD 89.6 ± 4.2 while in group C 81 – 90 cm 8(40%), 91 – 100 cm 1(5%), ≥ 101 cm 11(55%) Mean ± SD 100.2 ± 13.3.

Thus in group A waist size small in comparison A and B ($p < 0.001$) A and C ($p < 0.001$)

Table 13. Distribution of patient according to their waist hip ratio in the study group (group A), control group I (group B) and control group II (group C)

Waist Hip ratio	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
≤ 0.90	14	46.7	2	8	3	15
0.91 – 0.95	13	43.3	12	48	6	30
0.96 – 1.00	3	10	8	32	1	5
≥ 1.1	0	0	3	12	10	50
Mean ± SD	0.91 ± 0.04		0.95 ± 0.04		0.98 ± 0.07	

Statistical significance calculated :-

Between A and B groups

df = 53

t value = 0.8

p value = > 0.3

Between A and C groups

df = 48

t value = 1.1

p value = > 0.2

This table shows that the mean ± SD of waist hip ratio in group A was 0.91 ± 0.04, in group B it was 0.95 ± 0.04 and in group C it was found to be 0.98 ± 0.07.

Their was no statistical significant in waist hip ratio between A and B group (p > 0.3) and A and C group (p > 0.2).

Table 14. Distribution of patient according to Cardio Vascular Disease in the study group (group A), control group I (group B) and control group II (group C)

Cardio Vascular Disease	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
Hypertension	10	33.3	14	56	13	65
CAD	4	13.3	6	24	6	30
DF	4	13.3	2	8	1	5
CVA	0	0	0	0	0	0

This table shows that in group A hypertension in 10(33.3%), CAD 4(13.3%), DF 4(13.3%). Group B hypertension in 14(56%), CAD 6(24%), DF 2(8%) while in group C hypertension in 13(65%), CAD 6(30%), DF 1(5%) and none had CVA.

Statistical significance calculated: -

	Between A and B group	Between A and C group
	df 53	df 48
Hypertension	t value = 1.86 p value < 0.05	t value = 2.24 p value < 0.02
CAD	t value = 1.07 p value > 0.3	t value = 1.47 p value > 0.1
DF	t value = 0.73 p value > 0.4	t value = 0.97 p value > 0.3

Thus there was statistical significant difference in Hypertension between A and B ($p < 0.05$) and A and C ($p < 0.02$) while no statistical difference CAD and DF.

Table 15. Distribution of patient according to ophthalmic complications in the study group (group A), control group I (group B) and control group II (group C)

Ophthalmic complications	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
Cataract	5	16.7	2	8	1	5
Retinopathy	14	46.7	6	24	6	30

Statistical significance calculated: -

	Between A and B group	Between A and C group
	df 53	df 48
Cataract	t value = 0.98 p value > 0.3	t value = 1.27 p value > 0.2
Retinopathy	t value = 1.80 p value > 0.1	t value = 1.20 p value > 0.2

This table shows that retinopathy is higher in group A but no statistical difference between A and B ($p > 0.1$), A and C ($p > 0.2$).

Table 16. Distribution of patient according to Neuropathy in the study group (group A), control group I (group B) and control group II (group C)

Neuropathy	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
Peripheral	15	50	5	20	3	15
Autonomic	8	26.4	2	8	2	10
Impotency	11	36.3	4	16	2	10

This table shows that in group A PNP 15(50%), ANP 8(26.4%), Impotency 11(36.3%). Group B PNP 5(20%), ANP 2(8%), Impotency 4(16%) while in group C PNP 3(15%), ANP 2(10%), Impotency 2(10%).

Statistical significance calculated: -

	Between A and B group	Between A and C group
	df 53	df 48
PNP	t value = 2.36	t value = 2.77
	p value < 0.02	p value < 0.01
ANP	t value = 1.86	t value = 1.45
	p value < 0.05	p value > 0.1
Impotency	t value = 1.92	t value = 2.12
	p value < 0.05	p value < 0.05

Thus Peripheral neuropathy (p value (A and B) < 0.02), (p value (A and C) < 0.01). ANP (p value (A and B) < 0.05) (p value (A and C) > 0.01) and Impotency (p value (A and B) < 0.05) (p value (A and C) < 0.05) are higher in group A.

Table 17. Distribution of patient according to Infection in the study group (group A), control group I (group B) and control group II (group C)

Infection	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
Pul. Tuberculosis	5	16.6	2	8	1	5
Skin infection	7	23.3	1	4	0	0
Fungal Infection	1	3.3	0	0	0	0

Statistical significance calculated: -

Between A and B

df = 53

t value = 0.97

p value > 0.3

Between A and C

df = 48

t value = 1.16

p value > 0.2

This table shows that in group A Pul. TB 5(16.6%), Skin Infection 7(23.3%), Fungal Infection 1(3.3%). Group B Pul. TB 2(8%), Skin Infection 1(4%), Fungal Infection none, while in group C Pul. TB 1(5%), Skin Infection and Fungal Infection is none.

There was no statistical significant difference in Pulmonary tuberculosis infection or other infections.

Table 18. Distribution of patient according to their HbA_1C in the study group (group A), control group I (group B) and control group II (group C)

HbA_1C	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
≤ 6	6	20	5	20	5	25
6 – 8	9	30	12	48	10	50
8 – 10	5	16.7	5	20	4	20
≥ 10	10	33.3	3	12	1	5
Mean ± SD	8.6 ± 2.23		7.5 ± 1.09		7.15 ± 2.94	

Statistical significance calculated: -

Between A and B

df = 53

t value = 0.29

p value > 0.7

Between A and C

df = 48

t value = 0.32

p value > 0.7

This table shows that in group A blood sugar control 50% patients have HbA_1c value > 8% but no statistical significant difference in their mean control of blood sugar between A and B (p > 0.7), A and C (p > 0.7).

Table 19. Distribution of patient according to fasting serum insulin in the study group (group A), control group I (group B) and control group II (group C)

Fasting Insulin	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
< 6 (lower)	17	56.7	4	16	6	30
6 – 27 (normal)	13	43.3	18	72	12	60
> 27 (higher)	0	0	3	12	2	10

Statistical significance calculated in fasting insulin < 6 μ IV ml

Between A and B

df = 53

t value = 3.18

p value < 0.01

Between A and C

df = 48

t value = 1.89

p value > 0.05

This table shows that in group A fasting serum insulin lower in 17(56.7%) normal 13(43.3%), group B lower 4(16%) normal 18(72%) above normal 3(12%) while in group C lower 6(30%) normal 12(60%) and above normal 2(10%).

Thus group A patient show lower fasting insulin than group B ($p < 0.01$) and group C ($p < 0.05$).

Table 20. Distribution of patient according to their serum anti insulin antibody in the study group (group A), control group I (group B) and control group II (group C)

Anti Insulin antibody	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
Normal	27	90	25	100	20	100
Higher	3	10	0	0	0	0

Statistical significance calculated in fasting insulin < 6 μ IV ml

Between A and B

df = 53

t value = 1.67

p value > 0.1

Between A and C

df = 48

t value = 1.74

p value > 0.1

This table shows that there is statistically non significant difference between A and B and A and C groups.

Table 21. Distribution of patient according to their C-Peptide level in the study group (group A), control group I (group B) and control group II (group C)

Fasting C-peptide	Group A (n=30)		Group B (n=5)		Group C (n=4)	
	No.	%	No.	%	No.	%
Lower	8	26.4	4	80	2	50
Normal	22	73.6	1	20	2	50
Higher	0	0	0	0	0	0

This table shows that in group A patient C peptide level are normal 22(73.6%) and lower 8(26.4%), this show that there is normal insulin secretion in this group patients.

Table 22. Distribution of patient according to Renal function in the study group (group A), control group I (group B) and control group II (group C)

Renal function	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
S.Creatinine >1.5 mg %	1	3.3	0	0	0	0
Urine Alb. > 20μg/min	16	53.3	10	40	10	50
U Al/Creatinine > 30 μg / mg of S.creatinine	14	46.7	9	36	10	50

Statistical significance calculated for U Al / Cr

Between A and B

df = 53

t value = 0.76

p value > 0.4

Between A and C

df = 48

t value = 0.23

p value > 0.8

This table shows that in group A serum higher in only one patient while nephropathy (micro albumin urea U Al / Cr > 30 μg / mg of S.creatinine) in 14(46%) in group B 9(36%) group C 10(50%) but no statistical significance

Table 23. Distribution of patient according to their total cholesterol in the study group (group A), control group I (group B) and control group II (group C)

Total Cholesterol mg%	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
< 200 mg%	25	83.3	21	84	15	75
≥ 200 mg%	5	16.7	4	16	5	25
Mean ± SD	156.97± 53.8		168.96± 31.4		172.5± 79.9	

Statistical significance calculated: -

Between A and B

df = 53

t value = 0.07

p value > 0.9

Between A and C

df = 48

t value = 0.73

p value > 0.4

This table shows that total serum cholesterol in group A < 200 mg% in 25(83.3%) ≥ 200 mg% 5(16.7%) and mean ± SD 156.97 ± 53.8. In group B < 200 mg% in 21(84%) ≥ 200 mg% 4(16%) and mean ± SD 168.96 ± 31.4 while in group C < 200 mg% in 15(75%) ≥ 200 mg% 5(25%) and mean ± SD 172.50 ± 79.9.

There was no statistical significant difference for lower and higher total cholesterol patient in group A and B (p > 0.9) A and C (p > 0.4).

Table 24. Distribution of patient according to their HDL cholesterol in the study group (group A), control group I (group B) and control group II (group C)

Cholesterol HDL mg%	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
< 40 mg%	17	56.7	12	48	10	50
≥ 40 mg%	13	43.3	13	52	10	50
Mean ± SD	38.27 ±14.01		45.2 ± 13.19		36.7± 12.82	

Statistical significance calculated: -

Between A and B

df = 53

t value = 0.65

p value > 0.5

Between A and C

df = 48

t value = 0.47

p value > 0.6

This table shows that HDL in group A < 40 mg% in 17(56.7%) ≥ 40 mg% 13(43.3%) and mean ± SD 38.27 ± 14.01. In group B < 40 mg% in 12(48%) ≥ 40 mg% 13(52%) and mean ± SD 45.2 ± 13.19 while in group C < 40 mg% in 10(50%) ≥ 40 mg% 10(50%) and mean ± SD 36.7 ± 12.82.

There was no statistical significant difference for lower or higher HDL patient in group A and B (p > 0.5) A and C (p > 0.6)

Table 25. Distribution of patient according to their LDL cholesterol in the study group (group A), control group I (group B) and control group II (group C)

Cholesterol LDL mg%	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
< 130 mg%	28	93.3	24	96	19	95
≥ 130 mg%	2	6.7	1	4	1	5
Mean ± SD	88.4 ± 34.45		88.16±24.17		81.17±28.23	

Statistical significance calculated: -

Between A and B

df = 53

t value = 0.44

p value > 0.6

Between A and C

df = 48

t value = 0.25

p value > 0.8

There was no statistical significant difference in lower and higher LDL cholesterol patient in group A and B ($p > 0.6$) A and C ($p > 0.8$).

Table 26. Distribution of patient according to their VLDL cholesterol in the study group (group A), control group I (group B) and control group II (group C)

Cholesterol VLDL mg%	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
< 40 mg%	18	60	17	68	12	60
≥ 40 mg%	12	40	8	32	8	40
Mean ± SD	40.1 ± 24.1		35.8 ± 21.9		46.9 ± 29.8	

Statistical significance calculated: -

Between A and B

df = 53

t value = 3.38

p value < 0.001

Between A and C

df = 48

t value = 2.89

p value < 0.01

There was statistical significant difference in lower and higher VLDL cholesterol patients in group A and B ($p < 0.001$) A and C ($p < 0.01$).

Table 27. Distribution of patient according to their S.Triglyceride level in the study group (group A), control group I (group B) and control group II (group C)

S. trig mg%	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
< 150 mg%	12	40	8	32	9	45
≥ 150 mg%	18	60	17	68	11	55
Mean ± SD	171.7± 60.41		201.96± 97.83		208.69± 140	

Statistical significance calculated: -

Between A and B

df = 53

t value = 0.62

p value > 0.5

Between A and C

df = 48

t value = 0.35

p value > 0.7

This table shows that S.Trig in group A < 150 mg% in 12(40%) ≥ 150 mg% 18(60%) and mean ± SD 171.7 ± 60.41. In group B < 150 mg% in 8(32%) ≥ 150 mg% 17(68%) and mean ± SD 201.96 ± 97.83

while in group C < 150 mg% in 9(45%) ≥ 150 mg% 11(55%) and mean ± SD 208.69 ± 140.

There was no statistical significant difference in lower and higher S.Trig level in patient of group A and B ($p > 0.5$) A and C ($p > 0.7$).

Table 28. Distribution of patient according to treatment given in the study group (group A), control group I (group B) and control group II (group C)

Treatment	Group A (n=30)		Group B (n=25)		Group C (n=20)	
	No.	%	No.	%	No.	%
OHA	23	76.7	25	100	25	100
OHA + INS	7	23.3	0	0	0	0

Statistical significance calculated: -

Between A and B

df = 53

t value = 2.84

p value < 0.01

Between A and C

df = 48

t value = 2.37

p value < 0.02

This table shows that in group A patient taking OHA 23(76.7%) OHA+INS 7(23.3) group B OHA 25(100%) group C OHA 20(100%) and there was no statistically significant difference, treatment wise between A and B group ($p < 0.01$) and A and C ($p < 0.02$). But most of the group A patient are on OHA (76.7) only.

Discussion

Discussion

Present study was carried out on 30 patients of LB Type – 2 Diabetes mellitus (BMI < 18.5) in the Department of Medicine M.L.B. College Jhansi. These patients were attending either general medicine OPD, Diabetic clinic OPD, Gynaecology and Obstetrics OPD, Tuberculosis and chest OPD, and patients admitted in wards. Out of these 30 patients 22 (72.6 percent) were male and 8 (27.4 percent) were female. The mean BMI of patients were 17.19 ± 1.03. The clinical anthropometric biochemical and hormonal value were consequent to evaluation at a point of time. This study was planned to meticulously follow up a set of typical patient with LB Type-2 Diabetes mellitus over a period of six consecutive months.

In the past poverty and malnutrition have been vicariously linked to explain the variable difference observed in the diabetics in India as compared to west. Unfortunately the review as far as adult onset diabetic were concerned were often presumptive rather than factual. In the present study it was observed that more than 76 percent of patients (Table VI) with LB Type-2 diabetes mellitus were from middle and upper socioeconomic status and more than 66 percent were literate (Table VII). In India middle and upper class constitute about 50 percent of the population and literacy about 55 percent and these type of LB Type-2 diabetes mellitus was probably the real representation of diabetes mellitus prevalent in such society.

Thus this LB Type-2 diabetes mellitus constitute a distinct entity in themselves.

In present study LB Type-2 diabetes mellitus patients were similar to other Type-2 diabetes mellitus in context of male female ratio (Table II p value > 0.5 normal body weight Type-2 diabetes mellitus and > 0.8 over body weight Type-2 diabetes mellitus) 3:1, have age of onset 47.7 ± 8.82 (Table III) as compared to normal body weight Type-2 diabetes mellitus 47 ± 7.4 p value > 0.9 and overweight Type-2 diabetes mellitus 44.75 ± 9.37 p value < 0.001 (younger age of onset). Total duration of illness was 4.8 ± 3.14 (Table IV) as compared to normal body weight Type-2 Diabetes Mellitus 3.8 ± 2.57 p value < 0.05 (lesser duration) and over body weight Type-2 Diabetes Mellitus 4.5 ± 8.14 p value > 0.5. Urban and rural residence ratio was 1:1. p value for both normal and over weight Type-2 Diabetes Mellitus >0.4. Socioeconomic status (Table VI), Literacy status (Table VII) p value for normal body weight Type-2 Diabetes Mellitus > 0.9 and over body weight Type-2 Diabetes Mellitus > 0.1, and occupation and habit wise there was similarity with other Type-2 Diabetes Mellitus, but having gross dissimilarity in clinical, anthropometric, biochemical and treatment wise.

In present study family history of Diabetes Mellitus could be ascertained in 30 percent (Table X), much higher than normal body weight Type-2 Diabetes Mellitus 12 percent, p value < 0.05 and over body weight Type-2 Diabetes Mellitus 5 percent, p value < 0.05. Higher incidence of Diabetes Mellitus was also noted in siblings 20 percent. Similar family history was also observed by A.K.Baliar Sinha and S.Das in Cuttack 16.6 percent, Samar Banerjee and

Uttam.K.Paul in Calcutta 20 percent, B.k.Sahy in Hyderabad 30 percent and Vijay Vishwanathan in Chennai 42 percent. Yet they testify that LB Type-2 Diabetes Mellitus runs in families and not a variant of Type-1 Diabetes Mellitus or MRDM.

In present study most of the LB Type-2 Diabetes Mellitus presented with classic presentation (Table XI) 70 percent which was unlike to normal weight Type-2 Diabetes Mellitus 96 percent with p value < 0.02 and over weight Type-2 Diabetes Mellitus 95 percent with p value < 0.05. These LB Type-2 Diabetes Mellitus patients have frequent complain of parasthesia and numbness 36.7 percent weakness 50 percent and infection 13 percent at time of diagnostic of diabetes. These presenting complain at diagnosis was similarly observed by Anant Nigam in Jaipur who observed classic presentation in 29 percent weakness and fatigue 23.9 percent, parasthesia 19.5 percent and visual disturbances in 13.1 percent.

The anthropometric data revealed that they were not only lean but also had very low subcutaneous fat, this habit is typical of such LB Type-2 Diabetes Mellitus. Central adiposity with altered waist, waist hip ratio has been claimed to sin qua non of Type-2 Diabetes Mellitus, but these LB Type-2 Diabetes Mellitus had much lower waist 77.7 ± 3.8 cm, than normal body weight Type-2 Diabetes Mellitus 86.6 ± 4.2 cm, p value < 0.001 and over weight Type-2 Diabetes Mellitus 100.2 ± 13.3 cm p value < 0.001 (Table XII). But there waist hip ratio 0.91 ± 0.04 were similar to normal body weight Type-2 Diabetes Mellitus 0.95 ± 0.04, p value > 0.3 and over body weight Type-2 Diabetes Mellitus 0.98 ± 0.07 p value > 0.2.

In present study hypertension was present in 33 percent of LB Type-2 Diabetes Mellitus (Table XIV) much lower than normal body weight Type-2 Diabetes Mellitus 56 percent p value < 0.05 and over body weight Diabetes Mellitus 65 percent p value < 0.02. Coronary artery disease also lower to 13.3 percent than normal body weight Type-2 Diabetes Mellitus 24 percent p value > 0.03 and over weight Type-2 Diabetes Mellitus 30 percent p value > 0.1. In other study hypertension was much lower A.K.Baliar Sinha and S.Das in Cuttack (Hypertension 4.4 percent and CAD 16 percent), Anant Nigam in Jaipur (Hypertension 8.9 percent and CAD 10.2 percent), and K.Kanan in Madurai (Hypertension 18 percent and CAD 4 percent).

In this study of LB Type-2 Diabetes Mellitus patients with peripheral neuropathy were 50 percent much higher than normal body weight Type-2 Diabetes Mellitus 20 percent p value < 0.02 and over weight Type-2 Diabetes Mellitus 15 percent p value < 0.01 (Table XVI). Similar observations were noted by A.K.Baliar Sinha and S.Das in Cuttack 49 percent, Anant Nigam in Jaipur 19.8 percent, Vijay Vishwanathan in Chennai 31.9 percent.

The present study also reveals higher incidence of Impotency 36.3 percent (in LB Type-2 Diabetes Mellitus) and Autonomic neuropathy 24.4 percent in LB Type-2 Diabetes Mellitus than normal body weight Type-2 Diabetes Mellitus p value < 0.05 and over weight Type-2 Diabetes Mellitus p value < 0.05. Thus in LB Type-2 Diabetes Mellitus patients Impotency was much higher (Table XVI).

In the present study prevalence of Pulmonary Tuberculosis 16.6 percent and Skin infections 23.3 percent, were higher than normal body weight Type-2 Diabetes Mellitus (Pulmonary

Tuberculosis 8 percent and Skin infections 4 percent) and over weight Type-2 Diabetes Mellitus (Pulmonary Tuberculosis 5 percent and none Skin infections) (Table XVII). Similar observations were noted by A.K.Baliar Sinha and S.Das from Cuttack (Pulmonary Tuberculosis 7.7 percent and Skin infections 18.7 percent) and Anant Nigam in Jaipur (Pulmonary Tuberculosis 14.9 percent).

In this study eye complications was higher in LB Type-2 Diabetes Mellitus, (Cataract 16.7 percent and Retinopathy 46.7 percent) than normal body weight Type-2 Diabetes Mellitus (Cataract 8 percent and Retinopathy 24 percent) and over body weight Type-2 Diabetes Mellitus (Cataract 5 percent and Retinopathy 30 percent).

In present study of LB Type-2 Diabetes Mellitus patients present with overt albumen urea was 53.3 percent (Table XXII) than normal body weight Type-2 diabetes mellitus 40 percent p value > 0.4 and over body weight Type-2 Diabetes Mellitus 50 percent p value > 0.8. Thus no statistical significant difference was observed.

In this study of LB Type-2 Diabetes Mellitus has much higher albumin urea than other study probably better method used for detection of micro albumin urea.

In present study LB Type-2 Diabetes Mellitus patient had HbA_1c 8.6 ± 2.23 (blood sugar moderately controlled) than normal body weight Type-2 Diabetes Mellitus 7.5 ± 1.09 p value > 0.7 and over body weight Type-2 Diabetes Mellitus 7.15 ± 2.94 p value > 0.7 (Table XVIII). Inspite of moderate hyper glycemia, none of LB Type-2 Diabetes Mellitus patient goes in ketoacidosis, this show that they have insulin reserve, which prevent ketoacidosis similar to other

Diabetes Mellitus. Similar observations were noted by A.K.Baliar Sinha and S.Das in Cuttack, and Anant Nigam in Jaipur.

This study also revealed that LB Type-2 Diabetes Mellitus have fasting Serum Insulin less than normal in 56 percent and C-peptide was also lower in 56.7 percent, which was usually normal in normal body weight Type-2 Diabetes Mellitus in 72 percent, and over body weight Type-2 Diabetes Mellitus 60 percent.

This study of LB Type-2 Diabetes Mellitus 3 patients (10 percent), show anti insulin antibody (AIA) which was not detected in any other group patients.

In this study total Serum cholesterol in LB Type-2 Diabetes Mellitus 156.97 mg percent ± 53.8 comparison to normal body weight Type-2 Diabetes Mellitus, 168.96 mg percent ± 31.4 and over body weight Type-2 Diabetes Mellitus 172.50 mg percent ± 79.9 (Table XXIII). Hypertriglyceridemia (defined as > 150 mg percent, Table XXVII) in 60 percent LB Type-2 Diabetes Mellitus (171.7 mg percent ± 60.41) as compared to normal body weight Type-2 68 percent (201.96 mg percent ± 97.83) and over body weight Type-2 Diabetes Mellitus 55 percent (208.69 mg percent ± 140).

Average value of HDL cholesterol was 38.27 mg percent ± 14.01 as compared to normal body weight Type-2 Diabetes Mellitus 45.2 mg percent ± 13.19 and over body weight 36.7 mg percent ± 12.82. In LB Type-2 LDL cholesterol (88.4 mg percent ± 34.45) and VLDL cholesterol (40.1 mg percent ± 24.15) were lower as compared to normal body weight and over body weight.

Similar observations were noted by A.K.Baliar Sinha and S.Das in Cuttack, Samar Banerjee and Uttam.K.Paul in Calcutta and Anant Nigam in Jaipur.

In present study most of the patients of LB Type-2 Diabetes Mellitus have well controlled blood sugar by oral hyperglycemic agent, in 76.7 percent and only 23.3 percent were on insulin, (Table XXVIII), while none in normal body weight and over body weight Type-2 Diabetes Mellitus were on insulin. Same observations were noted by A.K.Baliar Sinha and S.Das in Cuttack (28 percent patients on insulin), B.K.Sahy in Hyderabad (12 percent patients on insulin) and K.Kanan in Madurai.

Conclusion

&

Summary

Conclusion and Summary

Overwhelming data from the present study of LB Type-2 diabetes mellitus established that LB Type-2 diabetes mellitus is a specific variant that fall into Type-2 diabetes mellitus. This is because the LB Type-2 diabetes mellitus patient have an adult age at onset, insidious presentation, are not nutritionally deprived, majority of them belong to middle and upper socio-economic class and not with a dominantly rural background and good literacy status. These patients have a positive family history, specially amongst sibling and have a substantial β cell reserve of insulin. They do not have autoimmune basis and most patients respond to oral hyperglycemic agents. These patients usually do not have a known secondary cause for diabetes mellitus.

Clinical presentation, complication, biochemical and hormonal profile are different for these patients in comparison to western Type-2 diabetes mellitus patients. There is low incidence of macrovascular disease in this group of patients and they have normal Serum cholesterol level in comparison to their western Type-2 diabetes mellitus counterparts. The study also established that peripheral neuropathy, weakness, impotency and infections are the presenting clinical feature in these patients. There exists insulin C-peptide disparity in the peripheral circulation due to excess utilization of insulin in the hepatic bed. These patients also show a clear-cut lack of hepatic insulin resistance as seen in patients with classic Type-2

diabetes mellitus. Hepatic function is hyperactive in these patients with excess of hepatic glucose output.

Similarity taken for this study were Male Female ratio, Socio-economic status, Literacy status, Residence, Habit, Age of onset and Total duration of diabetes.

Salient differentiating features between Low body weight, Normal body weight and Over body weight Type-2 diabetes mellitus.

Sl. No	Feature	LB Type-2 DM	Normal Body Weight Type-2 DM	Over Body Weight Type-2 DM
1.	Family History	High (30%)	Low (12%)	Very low (5%)
2.	Complain at diagnosis			
	a) Classic	70%	96%	95%
	b) Parasthesia and numbness	36.7%	16%	5%
	c) Weakness	50%	16%	10%
	d) Infection	13%	4%	0%
3.	Waist size	Low	Moderate	High
4.	CAD and Systemic HT	Low	High	Markedly High
5.	Neuropathy			
	a) Peripheral	High	Low	Low
	b) Autonomic	High	Low	Low
	c) Impotency	High	Very Low	Very Low
6.	Lipid profile			
	a) Total serum chol.	Normal	HighNormal	HighNormal
	b) S.Trig	High	Moderately High	Moderately High
7.	Treatment			
	a) OHA	76.7%	100%	100%
	b) Insulin	23.3%	0%	0%

The study therefore demonstrate that these LB Type-2 diabetes mellitus patients form a definite entity by themselves, having certain clinical, metabolic, hormonal as well as genetic distinction. This clinical entity needs to be recognized early to improve their metabolic control and thereby their quality of life.

Bibliography

Bibliography

1. Leo P. Krall, Rachmiel Levine, Donald Barnett. The history of diabetes Joslin's diabetes mellitus 13th edt.. 1-2.
2. Prof. Shigeaki Baba. Diabetes in new millenium edited by John R Turtle – VII.
3. WHO Diabetes mellitus Report of a WHO study group Geneva World Health Organization 1985 Tech Rep. Ser. No. 727.
4. National diabetes Data group classification and diagnosis of diabetes mellitus and other categories of glucose intolerance diabetes 1979 :28:1039 – 57.
5. Report of the expert committee on the diagnosis and classification of diabetes mellitus diab. Care 1997 :20; 1183 –97.
6. Alberiti KGMM and Zimmet P definition, diagnosis and classification of diabetes mellitus and its complication part-I. Diagnosis and classification of diabetes mellitus provisional report of WHO consultation diab Med. 1998 : 15 : 539 –53.
7. American diabetic association diabetic care 23 suppl – 1: 2000.
8. Mokdad AH, Ford ES, Bowman BA et al diabetic trends in the US 1990 –1998 diabetic care 23 : 1278 – 1283 :2000.
9. WHO prevention of diabetes mellitus report of WHO study group Geneva WHO 1994 Tech Rep. Ser. No. 844.
10. Mohan V. Vijaya Prabha R., Roma M et al clinical profile of lean NIDDM is South India Diab Res. Clinical pract 1997 ; 38:101 –108.

11. Hokstra J.B.L., Van Rijn H.J.M., Erkelens D.W. et al C-peptide diab care 1982 ; 5:438 – 46.

12. Ramchandran A., Jail M.V., Mohan V. et al high prevalence of diabetics in an urban population in South India Br. Med. J. 1988 – 297 – 90.

13. Das S. Low body weight Type-2 DM J. Nutr. Env. Med. 1999 : 90 : 235- 45.

14. Tripathy B.B. and Kar B.C. observation and clinical patterns of diabetes mellitus in India diabetes 1965, 14 : 4 : 9 – 12.

15. Malnutrition of diabetes in the tropics Report of the International workshop on type of diabetes peculiar to tropics diabetes care 1996 ; 19 :1014 – 17.

16. Nigam A. Lean NIDDM a definite entity in Dass Ed. Brochure on problem, practical aspect publication and questionaire. International workshop on type of diabetics peculiar to tropics 1995, Cuttack 1995 ; 54 –56.

17. Das S., Tripathy B.B., Samal K.C. et al plasma lipids and lipoprotein cholesterol in undernourished diabetic subjects and adults with protein energy malnutrition. Diabetes care 1984, 7 : 579 – 86.

18. Samal K.C., Das S., Agarwal B.N. et al Nutritional status and profile of NIDDM of recent onset. J diabetes Asso. Ind 1988; 28 : 99 – 101.

19. Das S., Mishra R.K., Jena B.B. et al Mortality amongst non insulin dependent diabetes mellitus patient in Orrisa. J. Asso. Phys. Ind. 1991 : 39 : 519 – 20.

20. Das S., Identity of lean NIDDM – clinical, metabolic and hormonal status. In : Kuchupillai N. Ed. Advances in Endocronology metabolism and diabetes vol –2 millinium India Ltd. Delhi 1994 : 42 – 53.

21. Das S., Samal K.C., and Tripathi B.B. metabolic and hormonal status in under weight NIDDM diabetes 1991 : 40 suppl – 1 : 34. A.

22. Das S., Reynold T, Patnaik A., et al plasma homocystemic concentration in Type-2 diabetic patient in India relation ship to body mass index J. diabetes compl. 1999.

23. Das S., Samal K.C., Baliar Sinha A.K. et al lean NIDDM pecularities and differences in metabolic and hormonal status J. Asso. Phys India 1995 – 43 : 392 – 42.

24. Das S., low body weight NIDDM an independent entity in Das A. K. ed. Medicine update vol 8 Association of physicians of India Mumbai 1998; 592 – 602.

25. Lathela J.R. insulin stimulated glucose metabolism liver structures and function. Acta Univaersalities ouluensis 1987 series D. Medica 1987 – No. – 162.

26. Nigam A. clinical and epidemological characteristics of NIDDM patient with low body weight diabetes 1997 16 (1) : 97 A.

27. Yagnic C.S., Shelgika K.M., Sardesai B.S., et al Association of obesity with clinical, biochemical, metabolic and endocrine measurement in newly diagnosed NIDDM patient in Nova Nardisk diabetes update proceddings 1993 ; Health care communications Bombay 1993 ; 139 – 46.

28. Mohan V., Deepa R., Bhatia E. et al Antibodies to pancreatic inlet cell antigens in diabetics seen in Southern India with particular reference to fibro calculus pancreatic diabetes Dia Med 1998 15 – 156 – 159.

29. Ram chandran A., Snehlata C, Satyavathi K. et al GAD 68 antibodies in classification of Asian India Diabetic subject with onset between 20 –40 yr. Int. J. Diabetes Dev. Cont. 1998 ; 18 suppl – 1 Abst No. 16.

30. Mohan V., Ramchandran A., and Vishwanathan M. Tropical diabetes in alberit KGMM and Krall L.P. Eds. Diabetes Annual vol Elseveir, Amsterdum 1986 – 30.

31. Kanan K. lean Type–2 diabetes mellitus – A distinct entity in Novo Nordisk diabetes update proceddings 1993 Health care communication Bombay 1993 ; 147 – 151.

32. Samal K.C., Das S., Parija C.R. et al C-peptide response to glycemic stemuli J. Asso. Phys India 1987 ; 35 : 362 – 402.

33. Das S., Mishra R.K., Samal K.C. et al Insulin and glycemic response to common carbohydrate diets in under nourished diabetes. J. Nutr. Med 1991 : 2 : 351 – 58.

34. Granner D.K. Hormones of the pancreas and gastro intestinal taract. In haropees biochemistry 24^{th-} Edn large medical publication standford 1996 – 586 – 96.

35. Davis S.N., Pagliassotti Mand, Cherrington A>D. liver metabolism in diabetes in marshal sm and HOME PD Eds. The diabetes Annual Vol 8. Elsevier Amsterdum 1994 ; 119 – 133.

36. Sotaneine E.A. and Karvonen I glucose load before and after enzyme including therapy in subject with glucose intolerance and

patient with NIDDM having hyper insulinemia or relative insulin deficiency diabetes Res 1989 ; 11 : 131 – 139.

37. Sahay B.K. profile of lean NIDDM as seen in Hyderabad in Novo Nordisk diabetes update proceedings 1993 health care communication Bombay 1993 ; 161 – 164.

38. Turner R., Strutlon I., Horton V. et al UKPDS 25 autoantibodies toislet cell cytoplasm and glutamic acid decarbohydrate for prediction of insulin requirement in Type- 2 diabetes lancet 1997 : 350, 1288 – 93.

39. Group L.C., Bottazzo G.F. and Doniach ICA identity latent Type-1 diabetes in patient aged 35 – 75 yrs. In diagnosis diabetes 1986, 35 : 237 – 41.

40. Tumori T., Carlsson T., Haiyan L., et al clinical and genetic characteristics of Type-2 diabetes with and without GAD antibodies diabetes 1999 ; 48 : 150 – 57.

41. Tumori T., Group L.C., Zinnet P.Z., et al antibodies to GAD reveal latent autoimmune diabetes mellitus in adults with non insulin dependent onset disease diabetes 1993 ; 42 : 359 – 62.

42. Kanungo A., Shtauvere A., Samal K.C., et al antibody to ICA 512 an GAD 65 in patient with NIDDM from Eastern India diabetologia 1997 ; 40 suppt – 1 Abst 164.

43. Dabadghao P., Bhatia E., Bhatia V., et al ICA in malnutrition related diabetes mellitus from North India diabetes res clinical pract. 1996 ; 34 : 73 – 78.

44. Irrine W.J., Sawers J.S., Feek C.M., et al the value of ICA in predicting secondary failure of oral hypoglycemia agent therapy in diabetics J. Clin. Lab Immunal 1979 – 2 – 23 – 26.

45. Thai A.C., N.G.W.Y., Loke K.Y., et al anti GAD antibodies in Chinese patient with youth and adult onset IDDM and NIDDM diabetologia 1997 : 40 : 1425 – 30.

46. Kobayashi T., Tanemoto K., Nokanishi K., et al immunogenetic and clinical characteristic and clinical characterisation of slowly progressive IDDM diabetes care 1993 : 16 ; 780 – 88.

47. Hagen C., Faber O.K., Binder C., Alberti KGMM lack of metabolic effect of C-peptide in normal subject and juvonil diabetic patient deta endocrinol 1977 – 85 suppl 209 – 257.

48. Mann J.I. and Lewis Barned N.J. dietary management of diabetes mellitus in North America In International text book of diabetes mellitus 2nd ed. John Wiley & Sons New York 1997 ; 759 – 71.

49. Baba S. Dietary management of diabetes mellitus in Japan In International text book of diabetes mellitus 2nd edn. John Wiley & Sons New York 1997 – 779 – 90.

50. Veena L.B. Psychological correlates of living with diabetes Phd. Thesis submitted to Andhra University Vishakapatnam 1997.

51. Sridhar G.R. & Madhu K. Prevalence of sleep disorder in diabetes mellitus diabetes res clin pract. 1994 : 23 : 183 – 86.

52. Sidharth Das, R.K. Mishra mortality events amongst NIDDM Ja p1 1991 vol 39 No. 7 : 519 – 20.

53. Mayes P.A. Glycolysis and oxidation of pyrovate and gluco neogenesis and control of blood glucose in harpers biochemistry, 24th edn. Lange medical publication stanford 1996 ; 177 and 201 – 202.

54. Sotaniemi E.A., Niemela O, Risteli L et al. Fibrotic process and

drug metabolism in alcoholic liver disease . Clin Pharmacol Therapeutics 1986;40:46-55.

55. Thurman R G and Kauffman F C. factors regulating drug Meta--bolism in intact hepatocytes. Pharmacol Rev 1980;31:229 – 51.

56. Chandalia HB, Sadikot S, Bhargava DK, and Krishnaswamy PR. Estimation of glycosylated haemoglobin by a simple chemical method and its use in monitoring control of diabetes mellitus. J Assoc Phys India 1980, 28: 285-6.

57. Brooks AP, Metcalfs J, Day JL and Edwards MS. Iron deficiency and glycosylated haemoglobin A_1 (letter). Lancet 1980; 2: 141.

58. Kumar S, Mangal BD, Alam SM, Vajpayee GM and Garg SK. A study of glycosylated haemoglobin in renal failure. J Assoc Phys India 1982; 31:69–71.

59. Franklin Bunn H. Evolution of glycosylated haemoglobin in diabetic patients. Diabetes 1981;30:613-7.

60. Hockstra JBL, Van Rijn HJM, Erkelens DW, Thijssen JHH. C-peptide. Diabetics care 1982; 5:438-46.

61. Field JB. Extraction of Insulin by liver. Ann Rev Med 1973; 24:309-14.

62. Hagen C, Faber OK, Binder C, Alberti KGMM. Lack of metabolic effect of C-peptide in normal subjects and juvenile diabetic patients. Acta Endocrinol 1977; 85(Suppl 209):25-7.

Master Chart

Master Chart

GROUP A. LOW BODY WEIGHT TYPE 2 D.M.(BMI < 18.5)

Sl. No.	Name	Sex	Age (yrs)	Age of Detection of Diabetes (yrs)	Total Duration of Illness (yrs)	Socio economic status	Residence	Complaint at diagnosis	Literacy status	Family History	Occupation	Habit	ANTHROPOMETRY								
													AT TIME OF PRESENTATION			AFTER 6 MONTHS OF FOLLOW UP					
													Wt (Kg)	Ht (mt)	BMI	Wt (Kg)	Ht (mt)	BMI	Waist (cm)	Hip (cm)	W / H
1.	Anar Kunwar	F	42	40	2	L	U	CL,C	IL	--	HW	V	38	1.44	18.32	40	1.44	19.29	66	74	0.89
2.	Munni Devi	F	46	44	2	M	R	CL	IL	--	HW	V	38	1.54	16.02	14	1.54	16.86	69	78	0.89
3.	Lalla	F	43	40	3	M	U	CL	IL	--	HW	NV	45	1.71	15.38	48	1.71	16.40	74	83	0.89
4.	Ram Kali	F	70	67	3	M	R	ES	IL	--	HW	V	38	1.44	18.32	38	1.44	18.32	70	80	0.78
5.	Kamlesh	F	46	42	4	M	R	CL,W	L	--	HW	V	45	1.75	14.60	48	1.75	15.67	64	72	0.88
6.	Dhayanwati	F	42	38	5	M	U	CL,P,W	IL	S,B	HW	V	40	1.48	18.25	40	1.48	18.25	68	74	0.91
7.	Durga	F	65	58	8	M	U	NN,P,W	IL	--	HW	V	44	1.56	18.08	44	1.56	18.08	72	76	0.94
8.	Kiran	F	67	36	12	M	U	CL	L	--	HW	V	47	1.60	18.36	50	1.60	19.53	76	84	0.90
9.	Bharat Bhusan	M	36	35	1	L	R	CL,W	L	--	F	V	52	1.70	17.99	52	1.70	17.99	74	83	0.89
10.	Umesh Chandra	M	53	52	1	M	R	DF,W	L	--	S	V	52	1.80	16.04	52	1.80	16.04	70	80	0.87
11.	Channa Ram	M	57	55	2	U	U	CL,W	L	FA	B	NV,A,S,TC	51	1.67	18.28	52	1.67	18.64	72	80	0.90
12.	Dhani Ram	M	72	70	2	L	R	NN	IL	--	F	NV,S,TC	46	1.60	17.96	46	1.60	17.96	68	72	0.94
13.	Vimal	M	42	40	2	M	R	W,P	L	--	F	NV	50	1.70	17.30	50	1.70	17.30	68	74	0.91
14.	Sukhlal	M	55	53	2	L	R	CL,N,P	L	--	F	NV,A,S,TC	42	1.60	16.40	46	1.60	17.96	68	72	0.94
15.	NarandraPalSingh	M	52	50	2	M	U	CL	L	M	S	NV,A,S,TC	45	1.67	16.30	48	1.67	17.20	70	76	0.92
16.	Shiv Shayasan	M	53	50	3	M	U	W,P	L	S	F	V,S,TC	43	1.58	17.20	45	1.58	18.20	72	80	0.90
17.	Manni Lal	M	60	57	3	M	U	FE,W,C	L	--	B	V	46	1.68	16.29	50	1.68	17.71	71	80	0.88
18.	Jagdish	M	46	42	4	M	R	CL	L	--	F	V	52	1.71	17.70	52	1.71	17.70	70	74	0.94
19.	Ramesh Chand	M	48	44	4	M	U	CL,W,NN	L	--	S	NV,A,S,TC	53	1.72	17.90	52	1.72	17.57	70	76	0.92
20.	Manni Ram	M	48	44	4	L	R	CL,W,P	L	--	B	NV,A,S,TC	43	1.58	17.44	45	1.57	18.25	74	76	0.97
21.	Girja Shankar	M	47	42	5	M	R	DF	L	--	S	NV,A,S,TC	48	1.62	18.28	48	1.62	18.28	74	80	0.92
22.	Bhayalal	M	48	43	5	M	R	CL,C,W	IL	FA	F	NV,A,S,TC	46	1.67	16.40	52	1.67	18.64	72	74	0.92
23.	Khairati	M	60	54	6	L	U	CL,W	L	B	B	V	54	1.76	17.40	54	1.76	17.43	74	84	0.88
24.	Pooran Namdev	M	39	33	6	L	R	CL	IL	--	F	V	46	1.59	18.19	46	1.59	18.19	73	76	0.96
25.	Raja Ram	M	53	47	6	M	U	CL	L	--	B	V	52	1.80	16.04	52	1.80	16.04	79	84	0.88
26.	Rajesh Kumar	M	63	55	8	M	R	CL,W,NN	L	B	S	NV,S,TC	46	1.66	16.68	48	1.66	17.41	68	76	0.89
27.	Prabhu Dayal	M	62	54	8	M	U	CL,W	L	--	F	NV,A,S,TC	50	1.73	16.70	50	1.73	16.71	79	82	0.96
28.	Baiz Nath	M	56	48	8	U	U	CL	L	B	B	V,TC	48	1.72	16.20	50	1.72	16.90	70	76	0.92
29.	Laxmi Narayan	M	59	48	11	U	U	W,C,FE,P	L	B	S	NV,A,S,TC	49	1.65	17.99	50	1.65	18.36	80	84	0.95
30.	Nathu Ram	M	62	50	12	M	R	CL	IL	--	F	NV,A,S,TC	44	1.58	17.62	45	1.58	18.22	76	82	0.92

GROUP A. LOW BODY WEIGHT TYPE 2 D.M. (BMI < 18.5)

SL. No.	COMPLICATION																	INVESTIGATION			
		CVD						RETINOPATHY		NEUROPATHY			INFECTIONS								
	KETOSIS	CAD	HT	CVA	PVD	DF	CATARACT	SIMPLE	PROLIFER--ATIVE	PNP	ANP	IMPOTENCY	PUL. TB	SKIN	FUNGAL	OTHERS	ECG	ECHO	C- X-RAY	OTHER FUNDAL CHANGES
1.	-	-	-	-	-	-	-	-	-	-	-	-	P	-	-	-	-	-	PT	-
2.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
4.	-	-	P	-	-	-	P	-	-	P	-	-	-	-	-	-	-	-	-	-
5.	-	-	-	-	-	-	-	-	-	-	P	-	P	-	-	-	-	-	PT	-
6.	-	-	-	-	-	-	-	-	-	P	-	-	-	P	-	-	-	-	-	-
7.	-	P	P	-	-	-	-	P	-	P	P	-	-	P	-	-	A	A	-	HCP
8.	-	-	-	-	-	-	-	P	-	P	P	-	-	-	-	-	-	-	-	-
9.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
10.	-	-	-	-	-	P	-	P	-	-	-	-	-	P	-	-	-	-	-	-
11.	-	-	-	-	-	-	P	P	-	-	-	-	-	-	-	-	-	-	-	-
12.	-	P	P	-	-	-	-	-	-	P	P	P	-	P	P	-	A	A	-	-
13.	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-
14.	-	-	-	-	-	-	-	-	-	P	P	P	P	-	-	-	-	-	PT	-
15.	-	-	-	-	-	-	-	P	-	P	-	P	-	P	-	-	-	-	-	-
16.	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-
17.	-	-	-	-	-	-	P	-	-	-	-	-	P	-	-	-	-	-	PT	-
18.	-	-	-	-	-	-	-	-	-	-	P	P	-	-	-	-	-	-	-	-
19.	-	-	-	-	-	-	-	-	-	P	-	P	-	-	-	-	-	-	-	-
20.	-	-	-	-	-	P	-	P	-	-	-	P	-	-	-	-	-	-	-	-
21.	-	-	P	-	-	P	-	P	-	P	-	-	-	-	-	-	-	-	-	-
22.	-	-	-	-	-	-	-	P	-	P	P	P	P	-	-	-	-	-	PT	-
23.	-	-	P	-	-	-	-	P	-	P	-	-	-	-	-	-	-	-	-	-
24.	-	-	-	-	-	-	-	-	-	-	-	P	-	P	-	-	-	-	-	-
25.	-	-	P	-	-	P	-	-	-	P	-	-	-	-	-	-	-	-	-	-
26.	-	P	P	-	-	-	P	P	-	P	-	P	-	-	-	-	A	A	-	HCP
27.	-	P	P	-	-	-	-	P	-	-	-	-	-	-	-	-	A	A	-	-
28.	-	-	-	-	-	-	-	P	-	P	-	-	-	-	-	-	-	-	-	-
29.	-	-	P	-	-	-	-	P	-	P	P	P	-	P	-	-	A	-	-	-
30.	-	-	P	-	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-

GROUP A. LOW BODY WEIGHT TYPE 2 D.M.(BMI < 18.5)

Sl. No-	BIOCHEMICAL EXAMINATION																		Treatment
									Lipid Profile (mg %)					Thyroid Function					
	FBG (Mg/dl)	HbA_1 C%	S.Insulin (µIU/ml)	AIA	C. PeP (ng/ml)	Urine Alb (µg/min)	S.Cr (mg%)	UAI/Cr (µg /mg of Cr.)	TCH	HDL	LDL	VLDL	S.Trig	T3 (ng/dl)	T4 (µg/dl)	TSH (µIU/ml)	AMA	
1.	280	11.0	2.2	7.2	< 0.5	> 60	0.8	> 60	87	17	54	115	213	104	6.4	1.9	NEG	OHA
2.	170	7.3	3.4	17.4	4.2	12.00	1.1	14.00	230	30	110	55	218	121	5.2	0.8	NEG	OHA+INS
3.	250	9.4	4.5	6.8	1.2	30.00	1.2	22.60	170	40	80	28	114	100	6.6	2.4	NEG	OHA
4.	133	6.0	11.8	6.1	1.5	> 60	0.9	> 60	130	44	65	23	192	112	7.3	0.7	$1:320^{-2}$	OHA
5.	243	9.9	9.6	5.2	0.7	48.15	0.8	32.18	264	53	174	37	185	159	11.3	1.6	NEG	OHA
6.	130	6.5	9.9	7.1	2.7	> 60	2.4	> 60	288	70	130	32	160	177	11.6	4.8	NEG	OHA
7.	240	11.0	16.0	7.2	2.5	> 60	1.0	> 60	148	26	85	40	220	130	8.4	0.6	NEG	OHA+INS
8.	173	7.8	9.7	7.2	< 0.5	12.71	0.8	18.30	74	22	52	16	79	162	11.8	1.6	NEG	OHA+INS
9.	480	> 17	6.5	7.6	2.1	10.48	1.5	15.54	130	42	66	27	135	112	6.9	1.8	NEG	OHA+INS
10.	183	8.1	< 2	5.2	< 0.5	35.60	1.0	30.10	184	34	95	38	274	128	7.5	1.4	NEG	OHA
11.	180	7.2	24	12	< 0.5	> 60	1.0	> 60	188	57	72	34	118	104	6.4	1.8	NEG	OHA
12.	80	5.0	7.1	8.6	4.1	> 60	1.6	> 60	116	26	60	20	140	153	11.2	1.5	NEG	OHA
13.	110	6.0	2	6.5	< 0.5	14.00	0.8	19.00	170	50	50	34	184	158	9.4	3.4	NEG	OHA
14.	370	13.7	3.9	5.6	0.8	16.16	1.0	23.31	183	32	114	37	187	96	6	2.9	NEG	OHA
15.	106	5.6	5.8	6.8	< 0.5	18.00	0.8	18.00	98	52	62	24	104	86	8.2	1.5	NEG	OHA
16.	70	4.7	2	6.1	< 0.5	35.00	0.9	50.46	154	52	50	42	208	106	6.6	1.3	NEG	OHA
17.	293	11.4	7.2	6.1	< 0.5	15.15	1.4	18.30	156	38	68	50	250	162	11.8	1.6	NEG	OHA
18.	283	11.0	20	7.8	< 0.5	17.20	1.1	24.85	143	14	85	45	224	135	8.4	0.7	NEG	OHA+INS
19.	280	11.0	2.2	7.2	< 0.5	> 60	0.8	>60	87	17	54	115	213	84	5.2	2.6	NEG	OHA
20.	190	8.3	5.6	7.6	1.2	> 60	1.0	> 60	109	33	65	26	133	103	7.2	1.7	NEG	OHA
21.	93	5.4	4.9	6.1	1.2	7.46	1.2	11.53	141	21	60	54	276	122	6.9	5.4	NEG	OHA
22.	207	8.8	5	7.1	< 0.5	38.50	1.0	55.43	106	52	53	22	112	119	7.9	1.0	NEG	OHA
23.	153	7.2	7.6	21.5	< 0.5	4.76	0.9	6.68	164	49	106	14	70	117	7.5	4.5	$1:320^{-2}$	OHA
24.	190	11.0	11.8	8.2	2	4.00	1.0	24.67	78	38	82	20	88	88	6.2	1.6	NEG	OHA
25.	238	10.6	3.4	18.7	2.5	> 60	1.8	18.40	246	28	105	58	168	150	11.5	1.8	$1:320^{-2}$	OHA+INS
26.	150	6.2	4.3	6.8	< 0.5	18.00	1.1	15.67	179	57	75	47	236	145	10.7	0.4	NEG	OHA
27.	363	10.5	10.6	5.6	< 0.5	> 60	1.2	> 60	168	52	90	58	130	153	9.3	0.4	NEG	OHA
28.	170	7.4	2.0	7.4	2	> 60	0.7	> 60	180	35	72	22	150	168	11.2	1.5	NEG	OHA+INS
29.	117	6.1	< 2	6.6	< 0.5	7.26	1.2	4.70	208	29	121	50	270	119	8.2	0.7	NEG	OHA
30.	120	6.6	5.4	7.2	2.1	14.00	1.0	16.20	130	38	80	20	100	143	7.2	1.0	NEG	OHA

GROUP B. NORMAL BODY WEIGHT TYPE 2 D.M. (BMI 18.5 - 25)

Sl. No.	Name	Sex	Age (yrs)	Age of Detection of Diabetes (yrs)	Total Duration of Illness (yrs)	Socio economic status	Residence	Complaint at diagnosis	Literacy status	Family History	Occupation	Habit	ANTHROPOMETRY								
													AT TIME OF PRESENTATION			AFTER 6 MONTHS OF FOLLOW UP			Waist (cm)	Hip (cm)	W / H
													Wt (Kg)	Ht (mt)	BMI	Wt (Kg)	Ht (mt)	BMI			
1.	Devki	F	55	54	1	L	R	CL,W	IL	--	HW	V	50	1.51	21.92	50	1.51	21.92	88	96	0.91
2.	Madhurima Singh	F	52	48	4	M	U	CL	L	--	HW	V	56	1.58	22.40	56	1.58	22.40	86	95	0.90
3.	Savitri	F	49	44	5	L	R	CL	IL	--	HW	V	60	1.63	22.58	64	1.63	24.00	90	96	0.93
4.	Parvati	F	74	67	7	M	U	CL,P	IL	--	HW	NV	45	1.42	22.30	48	1.42	23.80	88	94	0.91
5.	Rani Garg	F	58	50	8	M	U	CL	L	--	HW	V	50	1.50	22.20	52	1.50	23.10	92	98	0.95
6.	Nisar Ahmed	M	45	44	1	M	U	CL,W	IL	--	B	NV	69	1.70	23.04	69	1.70	23.80	95	89	1.06
7.	Kundan	M	44	43	1	L	R	DF	IL	--	F	NV,A,S,TC	57	1.57	24.10	57	1.57	24.10	87	92	0.94
8.	Shiv Kumar	M	48	47	1	L	R	CLP,NN	L	--	F	V	65	1.63	24.60	65	1.63	24.60	94	94	1.00
9.	Ram Sevak Yadav	M	52	50	2	M	R	CL	L	--	F	NV,A,S,TC	60	1.69	21.00	62	1.69	21.70	91	96	0.94
10.	S. N. Singh	M	48	46	2	M	U	CL	L	--	S	NV,A,S,TC	72	1.72	24.33	72	1.72	24.33	97	98	0.98
11.	S. P. Singh	M	56	54	2	M	U	CL	L	M	S	V	68	1.66	24.67	70	1.66	25.40	94	96	0.97
12.	Damodar	M	52	50	2	L	R	CL,FE,C	IL	--	F	NV,A,S,TC	72	1.76	23.20	72	1.76	23.20	95	98	0.96
13.	Ramashankar	M	36	34	2	L	R	CL	L	--	F	NV,A,S,TC	62	1.70	21.45	62	1.72	21.45	90	95	0.94
14.	S.B. Chaturvedi	M	58	55	3	M	U	CL	L	--	S	V,TC	65	1.68	23.00	67	1.68	23.73	88	96	0.91
15.	Baboo Lal	M	55	52	3	L	R	CL,W	IL	--	B	NV,A,S,TC	58	1.58	23.20	58	1.58	23.20	93	95	0.97
16.	Shri Ram	M	37	34	3	M	R	CL	L	FA,B	F	NV,A,S,TC	65	1.80	20.60	65	1.80	20.60	84	83	1.01
17.	Jagar Nath	M	52	49	3	L	R	CL,W	IL	--	F	V,TC	67	1.70	22.70	67	1.70	22.60	93	94	0.98
18.	A.K. Bhatt	M	56	53	3	U	U	CL	L	--	S	NV,A,S,TC	69	1.71	23.50	73	1.71	24.96	94	92	1.02
19.	S. Franklin	M	52	48	4	U	U	CL	L	B	S	NV,S,TC	70	1.68	24.80	70	1.68	24.80	92	95	0.96
20.	Shreyans	M	51	46	5	M	U	CL	L	--	S	V	62	1.70	21.43	62	1.70	21.43	82	88	0.93
21.	Ramesh Prasad	M	38	33	5	M	U	CL	L	--	S	V	55	1.67	19.72	55	1.67	19.72	86	96	0.89
22.	Pramod	M	48	43	5	M	U	CL	L	--	S	NV,TC	67	1.64	24.90	67	1.64	24.90	88	90	0.97
23.	S.P. Katiyar	M	50	45	5	U	U	CL	L	--	S	V	58	1.67	20.79	58	1.67	20.79	86	92	0.93
24.	Devi Deen	M	54	48	6	M	U	CL,P	L	--	B	NV,A,S,TC	53	1.65	19.46	53	1.65	19.46	84	88	0.95
25.	Sita Ram	M	50	38	12	M	U	CL	L	--	S	V	67	1.74	22.12	67	1.74	22.12	84	88	0.95

GROUP B. NORMAL BODY WEIGHT TYPE 2 D.M. (BMI 18.5 - 25)

SL. No.	COMPLICATION																	INVESTIGATION			
	KETOSIS	CVD		CVA	PVD	DF	CATARACT	RETINOPATHY		NEUROPATHY			INFECTIONS			OTHERS	ECG	ECHO	C- X-RAY	OTHER FUNDAL CHANGES
		CAD	HT					SIMPLE	PROLIFER--ATIVE	PNP	ANP	IMPOTENCY	PUL. TB	SKIN	FUNGAL					
1.	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-
2.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3.	-	-	P	-	-	-	-	P	-	-	-	-	-	-	-	-	-	-	-	HCP
4.	-	P	P	-	-	-	P	-	-	P	-	-	-	-	-	-	A	A	-	HCP
5.	-	-	P	-	-	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-
6.	-	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A	-	-
7.	-	-	P	-	-	P	-	-	-	P	-	-	-	-	-	-	-	-	-	-
8.	-	P	P	-	-	-	-	-	-	P	-	-	-	-	-	-	A	-	-	-
9.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
10.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
11.	-	P	P	-	-	-	-	-	-	-	P	P	-	-	-	-	A	-	-	HCP
12.	-	-	-	-	-	-	-	-	-	-	-	-	P	-	-	-	-	-	PT	-
13.	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	HCP
14.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
15.	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
16.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
17.	-	-	-	-	-	-	-	P	-	-	-	-	-	P	-	-	-	-	-	-
18.	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
19.	-	P	P	-	-	-	-	P	-	-	P	P	-	-	-	-	A	A	-	HCP
20.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
21.	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-
22.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
23.	-	-	P	-	-	-	-	P	-	-	-	P	-	-	-	-	-	-	-	-
24.	-	P	P	-	-	-	-	-	-	P	-	P	P	-	-	-	A	A	PT	HCP
25.	-	-	P	-	-	-	-	P	-	P	-	-	-	-	-	-	-	-	-	-

GROUP B. NORMAL BODY WEIGHT TYPE 2 D.M. (BMI 18.5 - 25)

Sl. No-	BIOCHEMICAL EXAMINATION																		Treatment
									Lipid Profile (mg %)					Thyroid Function					
	FBG (Mg/dl)	HbA_1 C%	S.Insulin (µIU/ml)	AIA	C. PeP (ng/ml)	Urine Alb (µg/min)	S.Cr (mg%)	UAI/Cr (µg /mg of Cr.)	TCH	HDL	LDL	VLDL	S.Trig	T3 (ng/dl)	T4 (µg/dl)	TSH (µIU/ml)	AMA	
1.	127	6.4	46.3	5.5	<0.5	9.04	0.8	14.20	161	30	87	73	363	129	8.4	0.36	NEG	OHA
2.	163	7.5	40.9	9.8	-	49.00	0.8	15.20	154	60	67	31	155	152	9.9	1.78	NEG	OHA
3.	107	5.8	14.4	7.3	-	9.50	0.8	15.01	176	47	102	3.8	138	173	12.3	5.29	NEG	OHA
4.	130	6.4	7.8	6.6	<0.5	38.00	1.0	>60.0	180	48	50	27	206	150	9.2	1.30	NEG	OHA
5.	184	8.2	22.0	9.3	-	28.00	0.9	42.40	210	52	88	38	156	78	8.0	0.80	NEG	OHA
6.	177	7.9	33.9	5.6	-	36.40	1.3	57.70	155	29	81	52	266	157	10.3	1.44	NEG	OHA
7.	137	6.7	9.9	5.3	<0.5	>60.0	1.0	>60.0	232	36	140	6.4	385	71	7.7	18.10	$1:320^2$	OHA
8.	136	6.5	24.6	5.8	-	9.80	0.8	18.00	172	36	80	68	184	110	8.2	8.80	NEG	OHA
9.	117	7.9	14.6	8.1	-	8.50	0.9	12.28	186	39	81	71	354	131	8.6	1.68	NEG	OHA
10.	77	4.9	17.4	6.2	-	9.49	1.1	14.11	223	32	116	3.6	469	174	12.3	2.20	NEG	OHA
11.	167	7.6	17.9	9.0	-	22.60	0.9	35.34	117	39	59	27	136	94	6.4	2.05	NEG	OHA
12.	380	12.4	8.6	5.8	-	11.10	1.0	18.20	152	44	74	68	138	102	8.4	1.70	NEG	OHA
13.	301	11.2	7.3	6.4	-	15.20	0.9	16.80	184	34	76	78	192	113	6.3	0.90	NEG	OHA
14.	103	5.7	10.7	8.5	<0.5	7.20	1.0	10.97	183	56	114	15	76	117	6.9	2.90	NEG	OHA
15.	110	6.5	9.8	5.3	-	8.20	1.2	12.00	150	50	106	28	188	138	5.2	1.60	NEG	OHA
16.	100	5.6	14.8	7.2	-	9.20	0.8	12.10	118	45	96	24	164	102	3.2	0.90	NEG	OHA
17.	172	7.8	23.4	5.2	1.1	18.20	1.0	18.20	160	48	116	50	138	76	8.2	2.40	NEG	OHA
18.	117	6.1	7.8	6.2	-	2.90	1.1	4.50	208	35	124	37	187	105	7.4	0.40	NEG	OHA
19.	183	8.1	<5.0	7.1	-	29.75	0.6	46.80	176	76	67	19	94	112	6.6	3.50	NEG	OHA
20.	148	7.2	12.5	8.2	-	12.50	0.9	12.50	164	68	58	27	184	88	12.2	2.40	NEG	OHA
21.	280	10.4	<5.0	7.8	-	18.60	1.0	22.40	150	72	82	27	136	88	3.6	0.80	NEG	OHA
22.	63	4.5	<5.0	6.4	-	14.10	0.9	22.09	178	38	106	37	186	115	6.9	3.00	NEG	OHA
23.	220	9.2	<5.0	7.0	-	>60.0	1.3	>60.0	141	51	63	40	200	126	8.8	1.90	NEG	OHA
24.	180	8.2	12.4	9.2	-	26.00	1.0	38.60	188	30	114	28	263	11	7.2	0.90	NEG	OHA
25.	197	8.9	6.2	6.0	-	>60.0	0.8	>60.0	106	35	57	18	91	94	4.4	0.30	NEG	OHA

GROUP C. OVER BODY WEIGHT TYPE 2 D.M. (BMI > 25)

Sl. No.	Name	Sex	Age (yrs)	Age of Detection of Diabetes (yrs)	Total Duration of Illness (yrs)	Socio economic status	Residence	Complaint at diagnosis	Literacy status	Family History	Occupation	Habit	ANTHROPOMETRY								
													AT TIME OF PRESENTATION			AFTER 6 MONTHS OF FOLLOW UP			Waist (cm)	Hip (cm)	W / H
													Wt (Kg)	Ht (mt)	BMI	Wt (Kg)	Ht (mt)	BMI			
1.	Ms. Chandrakani	F	40	38	2	M	U	CL,W	L	--	HW	V	82	1.66	29.70	86	1.66	31.20	96	102	0.94
2.	Usha Gupta	F	46	43	3	M	R	CL	L	--	HW	V	55	1.47	25.40	55	1.47	25.45	85	96	0.88
3.	Sultan Jahan	F	51	47	3	U	U	CL	IL	--	HW	NV	114	1.63	42.90	118	1.63	44.41	126	120	1.05
4.	Shanti	F	40	33	7	M	R	CL	IL	--	HW	V	73	1.52	31.50	72	1.52	31.50	108	102	1.05
5.	Karuna Srivastav	F	52	43	9	M	U	CL	L	--	HW	V	51	1.41	25.65	58	1.41	24.14	88	96	0.91
6.	Kiran Agarwal	F	40	30	11	M	U	CL	L	--	HW	V	64	1.56	26.29	64	1.56	26.29	84	94	0.89
7.	Prabhu Dayal	M	43	42	1	M	R	PLE	L	--	B	NV,A,S,TC	85	1.65	31.20	85	1.65	31.22	102	104	0.98
8.	Samshad Khan	M	40	39	1	L	R	CL,P,W	L	--	F	NV	73	1.70	25.25	73	1.70	25.25	88	94	0.93
9.	Ajay Kumar	M	50	49	1	M	R	CL	L	--	F	V	76	1.64	28.75	76	1.64	28.70	88	96	0.91
10.	Nasir Begh	M	50	48	2	L	R	CL	IL	--	F	NV	85	1.73	28.40	82	1.73	27.30	107	102	1.04
11.	Ajay Sharma	M	48	46	2	M	U	CL	L	FA	B	V	74	1.64	28.20	77	1.64	28.62	88	94	0.93
12.	Kailash	M	42	40	2	M	R	CL	L	--	F	NV,A,S,TC	62	1.51	27.19	66	1.51	28.90	101	100	1.01
13.	R. K. Saxena	M	50	48	2	M	U	CL	L	--	S	NV,A,S,TC	75	1.70	25.90	78	1.70	26.98	110	101	1.08
14.	Lovleen Sharma	M	39	36	3	U	U	CL	L	--	S	V	72	1.68	25.50	72	1.68	25.50	86	94	0.91
15.	Zafar Alam	M	52	47	5	U	U	CL	L	--	B	NV,S,TC	82	1.62	31.24	85	1.62	32.38	118	110	1.07
16.	G.P. Ojha	M	58	53	5	U	U	CL	L	--	B	NV,A,S,TC	100	1.78	31.60	100	1.78	31.60	123	112	1.09
17.	F.L Deruz	M	68	62	6	M	U	CL	L	--	R	NV,A,S,TC	85	1.73	28.40	87	1.73	29.00	110	104	1.05
18.	P.R. Mishra	M	76	68	8	M	U	CL	L	--	R	V,TC	86	1.74	28.40	83	1.74	27.41	103	100	1.03
19.	Arun Kumar	M	58	50	8	M	U	CL	L	--	S	NV,A,S,TC	75	1.65	27.50	75	1.65	27.80	86	94	0.89
20.	Harish Chandra	M	43	33	12	M	R	CL	L	--	F	V	88	1.74	29.00	88	1.74	29.00	107	102	1.04

F : FEMALE, M : MALE, SOCIOECONOMIC STATUS = L : LOWER, M : MIDDLE, U : UPPER
R : RURAL, U: URBAN, CL : CLASSIC (POLYUREA, POLYDYPSIA, WEIGHT LOSS), W : WEAKNESS, NN : NUMBNESS, P : PARSTHISA, FE : FEVER,
C : COUGH, L : LITERATE, IL : ILLITERATE, FA : FATHER, M : MOTHER, B: BROTHER, S : SISTER, HW : HOUSE WIFE, B : BUSINESS, F : FARMER,
S: SERVICE, R : RETIRED, L : LABOUR, V : VEGETARIAN, NV : NON-VEGETARIAN, A : ALCOHOLIC, S : SMOKER, TC : TOBACCO CHEWER

GROUP C. OVER BODY WEIGHT TYPE 2 D.M. (BMI > 25)

SL. No.	COMPLICATION																INVESTIGATION			
	KETOSIS	CVD		CVA	PVD	DF	CATARACT	RETINOPATHY		NEUROPATHY			INFECTIONS			OTHERS	ECG	ECHO	C- X-RAY	OTHER FUNDAL CHANGES
		CAD	HT					SIMPLE	PROLIFER--ATIVE	PNP	ANP	IMPOTENCY	PUL. TB	SKIN	FUNGAL					
1.	-	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	A	-	-	-
2.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3.	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
4.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
5.	-	-	P	-	-	-	-	P	-	-	-	-	-	-	-	-	-	-	-	HCP
6.	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-
7.	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	HCP
8.	-	-	P	-	-	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-
9.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
10.	-	-	-	-	-	P	-	-	-	-	P	P	-	-	-	-	-	-	-	-
11.	-	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A	-	-
12.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
13.	-	-	-	-	-	-	-	-	-	-	P	P	-	-	-	-	-	-	-	-
14.	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
15.	-	-	P	-	-	-	-	P	-	P	-	-	-	-	-	-	-	-	-	-
16.	-	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A	-	HCP
17.	-	P	P	-	-	-	-	P	-	P	-	-	-	-	-	-	A	-	-	-
18.	-	P	P	-	-	-	P	-	-	-	-	-	-	-	-	-	A	A	-	HCP
19.	-	P	P	-	-	-	P	P	-	-	-	-	P	-	-	-	A	A	-	-
20.	-	-	P	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	PT	HCP

CVD : CARDIO VASCULAR DISEASE. CAD : CORNORY ARTERY DISEASE. HT : HYPER TENSION. CVA : CEREBRO VASCULAR DISEASE PVD : PERIPHERAL VASCULAR DISEASE. DF : DIABETIC FOOT. PNP : PERIPHERAL NEUROPATHY. ANP : AUTONOMIC NEUROPATHY. PUL.TB : PULMONARY TUBERCULOSIS. P : PRESENT. A : ABNORMAL. HCP : HYPERTENSIVE CHANGES PRESENT.

GROUP C. OVER BODY WEIGHT TYPE 2 D.M. (BMI > 25)

Sl. No-	BIOCHEMICAL EXAMINATION																	Treatment
									Lipid Profile (mg %)					Thyroid Function				
	FBG (Mg/dl)	HbA_1C%	S.Insulin (µIU/ml)	AIA	C. PeP (ng/ml)	Urine Alb (µg/min)	S.Cr (mg%)	UAI/Cr (µg /mg of Cr.)	TCH	HDL	LDL	VLDL	S.Trig	T3 (ng/dl)	T4 (µg/dl)	TSH (µIU/ml)	AMA	
1.	150	7.1	<2.0	7.5	-	7.25	0.7	11.60	130	46	58	26	130	142	8.9	3.1	NEG	OHA
2.	83	5.1	5.4	8.7	-	26.53	0.8	39.30	153	11	92	72	359	120	8.6	1.9	NEG	OHA
3.	205	8.6	18.0	7.3	-	18.00	0.9	25.90	170	54	72	38	168	112	10.4	2.4	NEG	OHA
4.	134	6.2	4.7	4.2	-	24.70	1.2	36.80	87	43	60	32	188	98	6.5	3.8	NEG	OHA
5.	200	7.8	12.8	8.7	-	28.20	1.0	40.60	158	50	98	42	138	116	12.0	1.2	NEG	OHA
6.	243	9.9	29.7	7.9	< 0.5	5.25	0.9	4.77	161	44	92	31	155	96	5.8	1.7	NEG	OHA+INS
7.	146	6.8	8.2	8.6	2.0	12.80	0.8	22.00	180	42	98	68	144	112	5.6	1.8	NEG	OHA
8.	138	5.7	13.4	6.4	-	35.20	0.8	50.68	260	22	86	26	222	130	8.6	0.7	NEG	OHA
9.	90	5.3	6.8	6.8	-	7.50	1.1	11.60	161	52	97	21	107	85	5.2	6.8	NEG	OHA
10.	188	7.4	11.5	3.8	-	12.11	1.0	17.28	97	44	70	32	130	112	11.2	0.4	NEG	OHA
11	320	11.2	3.2	6.4	-	26.60	1.3	39.80	270	24	118	56	212	87	6.6	2.7	NEG	OHA
12	150	6.5	18.2	8.0	-	12.20	0.8	18.60	170	32	92	52	148	66	5.4	4.6	NEG	OHA
13.	140	6.6	5.8	8.1	-	22.70	1.0	32.70	135	27	69	39	194	15	9.6	5.0	NEG	OHA
14.	120	6.2	14.1	7.0	1.6	9.30	1.2	14.52	242	51	147	48	240	127	7.7	2.0	NEG	OHA
15.	283	9.8	8.4	9.3	-	8.30	1.0	11.60	205	24	84	64	192	118	8.2	0.6	NEG	OHA
16.	192	8.4	11.6	8.4	-	26.20	0.9	32.40	280	32	118	70	340	114	4.2	2.8	NEG	OHA
17.	167	7.6	60.0	6.5	-	42.35	1.4	>60.0	191	26	96	138	692	67	0.9	3.6	NEG	OHA
18.	83	5.1	3.9	6.7	-	17.65	1.1	11.98	127	32	70	27	135	173	12.0	3.7	NEG	OHA
19.	150	6.5	8.3	9.6	-	>60.0	1.0	>60.0	111	34	59	26	130	88	5.7	1.6	NEG	OHA
20.	100	5.6	8.8	5.2	< 0.5	>60.0	0.7	>60.0	162	44	92	30	149	139	10.0	1.6	NEG	OHA

NORMAL VALUES :-

FBG : 90 – 120 mg/dl, HbA_1c % (< 6), INSULIN : 6 – 27 (µIU/ml), AIA : ANTI INSULIN ANTIBODY (< 10 %), C-PEPTIDE : 1.1 – 5.0 (ng/ml), URINE ALB : < 20 (µg/min), S.Cr : (0.7 – 1.5 mg %), UAI/Cr (< 30µg/mg of Cr.), TCH : 125 – 200mg%, HDL : 30 – 65mg%, LDL : 85 – 130mg%, VLDL : 5 – 40 mg%, S.Trig : 85 – 130mg%, T3 : 60 – 180 (ng/dl), T4 : 3.2 – 12.60 (µg/dl), TSH : (0.3 – 5.5 µIU/ml), AMA(ANTI MICROSOMAL(Anti TPO) ANTIBODY) : Negative < $1:10^2$, Positive > $1:10^2$, OHA : ORAL HYPOGLYCEMIC AGENT, INS : INSULIN.